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SUBJECTIVE VISUAL SENSATIONS

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Having been subject to distressing subjective visual sensations during the greater part of my life at intervals varying from three or four days to a month or even longer, I think it is worth while to describe them in detail. I have made numerous notes from time to time and have recorded the effects of various remedies which I have tried in order to obtain relief.

Unfortunately I have had no opportunity of consulting the various books and medical journals, as there is no medical library in Durban, where I reside. However, as I have taken a large number of notes at the time of the attacks, a report may be of some interest and value.

The sensations always take the form of what is commonly called fortification spectra or teichopsia and have never in my case been accompanied with migraine or sick headache. Moreover I have never had any decayed teeth or nasal or pharyngeal obstruction, nor have I suffered from any optical defect or eyestrain. In fact, the only causes to which I can attribute the attacks are anemia and occasional attacks of dizziness, which pass away quickly. My blood pressure is decidedly subnormal; my heart beat and pulse rate are normal and regular, and my arteries are fairly soft.

I must have had several hundred attacks of these visual sensations. They begin without the slightest warning or premonitory symptoms. They never occur when I am in bed but generally take place between meals. Frequently an attack comes on when I am reading. Then I suddenly discover that I am unable to read distinctly with either eye, and I notice a quivering in the air directly in front of me. This usually begins when I have been looking at a white surface or a bright sky. Sometimes it commences when I have been looking through a microscope, whether illuminated by artificial light or by daylight. Then if I look at anything, especially a printed page, I notice that the object or print appears blurred and tremulous. I have frequently made a chart of my fields of vision with the perimeter, but I have never found them to be more contracted than after the symptoms have disappeared. Although the general visual fields are unaffected, the macular fields become exceedingly contracted. Each normal macular field when I am reading print at 10 inches (25 cm.) is an ellipse measuring 25 mm. in the horizontal axis and about 20 mm. in the vertical axis. The area becomes contracted to about 5 mm. in the vertical and 3 mm. in the horizontal axis.

The normal area of critical vision is represented by a large oval. No letter can be clearly seen outside it unless the eyes move slightly. A small oval

represents the contracted area of critical vision during an attack. I have found it better to try the experiment with one eye shut, as otherwise the eyes unconsciously wander away from the fixation letter. Owing to this contraction of the macular field I find it next to impossible to read the words, as I must pick them out one by one, and that only increases the disturbance. Moreover the accommodation becomes greatly weakened, so that only distant objects can be clearly seen, and then only with a feeling of strain.

At the same time the pupils become widely dilated, which confuses my distant vision, as my eyes are myopic (6 diopters) and I continually wear glasses that are slightly undercorrected, as I cannot bear to wear the full correction, a condition very often met with in cases of a high degree of myopia. It is curious that the general field of vision is never contracted either for white or for colors. I may add that the visual field for each eye is abnormally large both for white and for colors, while the vision when corrected is slightly above the normal. I have on many occasions when suffering from an attack had my eyes examined by eminent oculists, but they have never been able to detect any abnormality either in the general appearance of the fundus or in the disk or any alteration in the circulation or caliber of the vessels.

After the contraction of the macular area has lasted for about a minute, or sometimes rather less, I begin to notice a general tremulous motion in everything I see, as though I were looking through a current of hot air or steam. This is immediately followed by the appearance of a zigzag C-shaped figure, the well known fortification spectrum. One spectrum usually appears in front of each eye, and the two are similar in all respects. They are admirably depicted in color in Dr. Liveings' classic work on migraine. My spectra are very similar to those that he has described, except that they do not as a rule show any colors except an intensely bright, almost dazzling gold, which flickers rapidly. The subjective figure is independent of vision; when I close my eyes tightly, I see it much more plainly as a brilliantly illuminated crescent in front of each eye against a black background.

Each of the two spectra forms a C-shaped figure, which as projected into space appears to be about 1 foot (30 cm.) in diameter, apparently much larger than a full moon. The gap in the spectrum seen in front of the left eye faces the nasal side, while that in front of the right eye faces the temporal side. But the figures frequently face the other way, i. e., with both gaps facing left. The zigzag serrations form two rows. Occasionally, three rows are seen, the inner one being exceedingly bright or incandescent. The center one is yellowish, like a gas flame, while the third one, when it is seen, has a slight tinge of red.

The spectra are always in rapid vibration, resembling a field of corn in a strong wind. The motion is always centrifugal and exceedingly rapid. I have made innumerable experiments to try to ascertain the rate of vibration, by comparing the motion with bodies set to vibrate at known velocities. As the result of many trials I have found the rate to be more than 8 and less than 12 vibrations a second.

After about twenty minutes, sometimes less and sometimes longer but never more than half an hour, the figures spread out and become fainter and fainter, until at length they finally vanish. The macular fields increase in area at the same time, until when the images disappear the vision simultaneously returns to normal.

As a rule, two images are seen, one before each eye, and they are identical as regards size, position and shape. It seems to me that there are only two places where such images can originate—at the fovea and at the chiasma, for I have

found that no movement of the eyes can alter the positions of the spectra or cause them to fuse, which naturally must be the case if the stimulus is situated at the chiasma.

Although the disturbance may be traced to the chiasma, it may be that it must be looked for at the fovea itself, or just behind it in the choroid. The fovea and the inner half of the macula contain no trace of blood vessels or even capillaries, the area being nourished by plasma only, since any blood corpuscles would interfere with critical vision. The fact, however, that the two C-shaped crescents are identical in every respect seems to me to point to the chiasma rather than to the maculae,

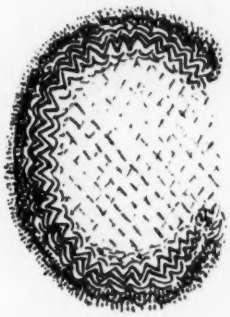

Fig. 1.—General appearance of the fortification spectrum. Drawn from life.

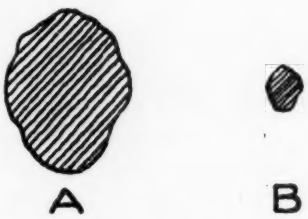

Fig. 2.—*A*, the normal area of critical vision seen at 8 inches (20 cm.). *B*, the contracted area during an attack.

for were the spectra to arise from the two maculae separately, why should they be absolutely the same in all respects? But in either case I think the affection must be traced to anemia dependent in some cases on atonic dyspepsia, affecting the central bundle of nerve fibers, which Prof. Uhthoff traced along the optic nerve to the chiasma. I think it is highly improbable that the origin of the spectra can be traced beyond the chiasma, where the fibers intertwine and partly cross over the optic tracts.

I have experimented with a large number of drugs to obtain relief from the disturbance, but none of them seems to have had much effect. Tincture of cannabis in 5 or 10 minim (0.3 or 0.6 cc.) doses is perhaps

the most effective. Also a cup of strong coffee with a tablespoonful of brandy seems to have a slight effect in hastening the disappearance of the spectra. But by far the most beneficial method that I have found has been to lie down flat and close my eyes or make the room dark. A bandage may be tied over the eyes until the images have vanished (they can be seen even in absolute darkness). If this is done as soon as the symptoms are noticed, the trouble should be all over in about a quarter of an hour.

I think that this disturbance may be attributed either to anemia or to atonic dyspepsia or more often to a combination of the two. When an attack is over I have no symptoms except slight dizziness or faintness, which rapidly disappears.

In conclusion, I wish to draw attention to the importance of plotting the area of critical vision in cases of real or suspected toxic amblyopia arising from any cause.

TRANSITORY WORD BLINDNESS ASSOCIATED WITH RIGHT HOMONYMOUS HEMIANOPIA

REPORT OF A CASE IN A PATIENT WITH CANCER OF THE PROSTATE GLAND

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Dyslexia, or word blindness, is a peculiar psychic disturbance in which the patient is unable to read in spite of normal vision. The defect may be either congenital or acquired. It is present congenitally in about 1 per cent of all persons. It is characterized by a disproportion between the intellectual development of the child and its reading ability, which is affected in varying degree. There may appear a total lack of comprehension of the reading matter. The subject frequently reverses the order of letters or words or may fail to understand certain words. These difficulties may be more pronounced when the child is trying to read fast. Such children are often misunderstood by their teachers, being looked on as obstinate or inattentive.

Acquired dyslexia (alexia, verbal visual agnosia) is also made manifest by the inability of the patient to understand written or printed language, even though the vision is good. As a rule he can understand spoken language without difficulty. The word blindness may be partial, so that the patient may retain, for example, the ability to read written but not printed words, figures but not letters, etc.

The onset of dyslexia may be very sudden. The etiology is diverse, but the pathologic changes are localized in the posterior portion of the outer, upper surface of the angular gyrus of the temporal lobe and the contiguous portions of the occipital lobe. The lesion may often give rise to contralateral homonymous hemianopia. In a pronounced left-handed person, disease localized to the right gyrus angularis, ceteris paribus, will give rise to a corresponding condition—left homonymous hemianopia. If the patient states that he is left-handed, yet acquires right homonymous hemianopia, it is reasonable to believe that he is not entirely left-handed by nature.

The case to be reported is of interest not only from the ophthalmologic, but from the psychologic, and even the philologic, point of view.

REPORT OF CASE

A man, aged 65, apparently healthy and strong, came to see me for the first time on May 15, 1935. His complaint dated from the previous April 17, at which time he first noticed a disturbance in his ability to read. On reading a letter

which he had dictated to his stenographer he was astonished to find that it contained numerous invectives. When read aloud to him by the stenographer, however, it sounded precisely as he had dictated it. The patient, immediately aware that something was wrong with his mental processes, tried to find certain well known names in the telephone directory. Some of these he could find and others he could not. His general health had always been good except that for the past

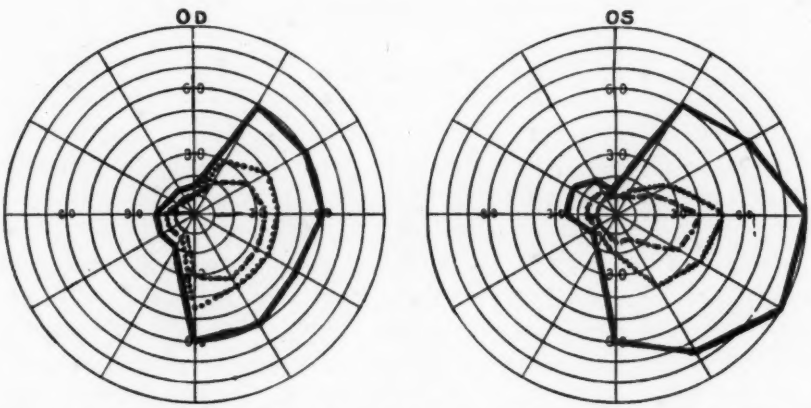

Chart 1.—Visual fields, showing right homonymous hemianopia. The solid line indicates the results of the examination with a 2 mm. white test object; the dotted line, the results with a 2 mm. red test object, and the dash and dot line, the results with a 2 mm. green test object.

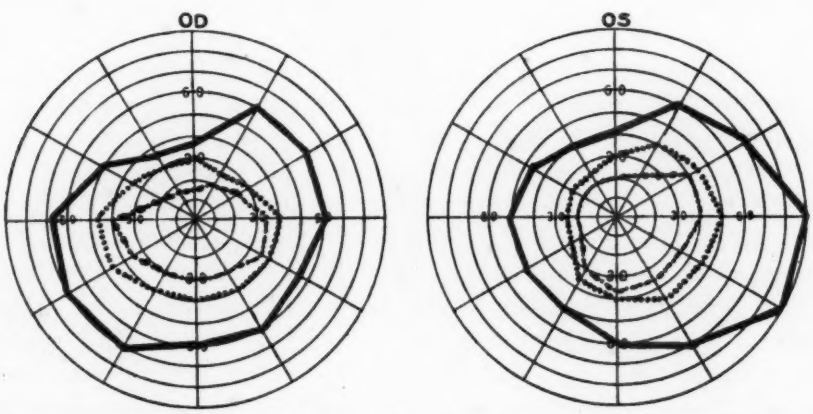

Chart 2.—Visual fields taken five weeks after those in chart 1, showing considerable improvement. The solid line indicates the results of the examination with a 2 mm. white test object; the dotted line, the results with a 10 mm. red test object, and the dot and dash line, the results with a 10 mm. green test object.

year he had had symptoms referable to prostatic hypertrophy. For these, he was now undergoing a series of roentgen treatments. He was left-handed.

Examination of the eyes revealed normal findings. The vision in the right eye with a + 1.50 sphere was 7/10, and that of the left eye with a + 2 sphere, 7/7. Examination of the visual fields showed a pronounced right homonymous hemianopia, especially for colors, more marked in the left eye (chart 1).

On reading the patient made many characteristic mistakes. He consistently read b for p, g for k, t for d and f for p. Some words he could not read at all, yet he understood them when they were read to him. There was no aphasia.

The prognosis was considered good, and potassium iodide was prescribed. Five weeks later the patient returned, obviously a doomed man. He was cachectic and had a bad color. The ocular changes remained the same except that the visual fields showed considerable improvement (chart 2). He was now able to read any text faultlessly. The blood pressure was found to be about 180 systolic and 100 diastolic. Examination of the urine did not reveal any albumin or sugar.

Two months later the patient died from prostatic cancer. As autopsy was not performed, it is impossible to state definitely whether the hemianopia and dyslexia were due to a carcinomatous metastasis or to cerebral vascular disease. The increase in blood pressure might point to the latter possibility.

In the literature to which I have had access I have found no case entirely similar to this one. To me, the interesting feature is the peculiar way in which the dyslexia manifested itself. It seems fair to assume that the patient had a certain amount of ill feeling toward the person to whom the dictated letter was addressed. Under ordinary circumstances this ill feeling was suppressed and hidden beneath conventional polite phraseology. The patient's acute cerebral disturbance, however, interfered with his appreciation of certain word pictures, whereupon he promptly substituted the word images of the invectives he had always been tempted to use:

Such a condition might give rise to legal problems, for instance: The patient writes a letter himself, by hand. He does not know anything about his own ailment, this having arisen shortly before. In his letter he writes invectives instead of polite phrases. Is he then juridically responsible for his blunder? Or the patient writes a letter to B. In his letter he mentions A, whom he abuses. In good faith B uses the expressions about A. Who, then, is the responsible party—the patient or B? The examples might be multiplied indefinitely.

Another peculiarity of the case reported here is that the patient in interchanging consonants strictly followed the so-called phonetic law of Verner. As will be recalled, Jacob Grimm, a German, enounced his phonetic law in 1822. In 1876 this was amplified by the Dane Karl Adolf Verner in his famous work "Die Konsonantverschiebungen der indogermanischen Sprachen." Starting with Sanscrit, Verner proved that there was a constant and regular consonant alteration traceable through the Greek, Latin, old Norwegian, German and Scandinavian languages. Thus *bhiter* in Sanscrit bacame πατὴρ in Greek, *pater* in Latin, *vater* in German, father in English and *fader* in the Scandinavian languages.

Finally I want to mention that the considerable improvement in the patient's fields of vision may have been due to the fact that he was partially left-handed. As the homonymous hemianopia was right-sided he could hardly have been absolutely left-handed.

RELATION BETWEEN BLUE SCLERAS AND HYPERPARATHYROIDISM

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The clinical entity consisting of blue scleras, osteoporosis with spontaneous fractures and deafness and the dominant hereditary transmission of the syndrome have long been known to ophthalmologists and pediatricians. The association of blue scleras with fragilitas ossium was noted by Ekman (1788), Lobstein (1833) and von Ammon. Van der Hoeve and de Kleyn [1] and Bronson [2] independently included hereditary deafness (which, according to some observers, may develop only after the age of 20) as a part of the entity. German authors commonly refer to this symptom complex as the van der Hoeve and de Kleyn syndrome, while in the English literature there is a tendency to link the entity with the name of Lobstein.

It is not pertinent to the purpose of this article to delve into the early historical facts or to decide to which authorities credit should be given for advancement in the knowledge of the syndrome of blue scleras. An excellent and exhaustive historical review was recently made by Dessoff.[3] In our search of the literature confined to data bearing on the etiology we found varied and conflicting theories, in which lies a potential danger too often overlooked. Conclusions loosely drawn or founded on insufficient facts may lead to treatment, even operative intervention, involving serious consequences.

In the Netherlands and England, where cases of the syndrome have been frequently reported, the explanation was generally made that the condition is due to a deficient development of the mesenchyma, since most of the organs affected are of mesenchymal origin (Eddowes, Peters, Freytag). In recent years many authors have considered that alteration in the ductless glands may be the causative factor (Behr, Bolten, Gutzeit, Hofmann); others, like Takahashi [4] and Kaznelson,[5]

1. van der Hoeve, J., and de Kleyn, A.: Arch. f. Ophth. **95**:80, 1918.
2. Bronson, E.: Edinburgh M. J. **18**:240, 1917.
3. Dessoff, J.: Blue Sclerotics, Fragile Bones and Deafness, Arch. Ophth. **12**:60 (July) 1934.
4. Takahashi, T.: Arch. f. Ophth. **115**:206, 1925.
5. Kaznelson: Zentralbl. f. d. ges. Ophth. **18**:710, 1918.

made an attempt to explain the syndrome as entirely caused by hypofunctioning of the parathyroid glands, involving hypocalcemia. In contradiction of this view, Dessoff pointed out the possibility that hyperparathyroidism might be the sole cause. The comparison and evaluation of known facts, in addition to further investigation, are essential to an understanding of this uncommon symptom complex. We have made a detailed study of 2 cases that came under our observation, with the purpose of throwing light on the relationship between the syndrome of blue scleras and that of hyperparathyroidism.

THE SYNDROME OF BLUE SCLERAS

A marked hereditary tendency is inherent in fragilitas ossium, a condition characterized by imperfectly generalized ossification of all the bones. In these cases the child may be born dead, showing multiple fractures acquired in utero, or these may not develop until soon after birth. The number of fractures is at times outstanding. A child 15 months old who came under our observation had eight fractures, according to a single roentgenogram of the lower extremities. In other instances children who are apparently normal at birth do not show evidence of brittleness of the bones in early infancy, but this develops after a delay, in later childhood or early adolescence. There is a tendency for the condition to become arrested after puberty and gradually recede or disappear as time passes. Boyd [6] mentioned, in addition to the foregoing characteristics, osseous swellings that sometimes occur in the occipital and frontal regions and especially in the temporal region, so that the ears are turned outward and down. Furthermore, owing to incomplete ossification, the skull appears as a mere membranous bag or a few osseous plates. If ossification proceeds somewhat further the skull may present a large number of wormian bones.

In exceptional cases the hereditary tendency may be absent, as in one case reported by Gutzeit,[7] but a study of the antecedents invariably shows the anomaly through many generations. Investigation into the family tree discloses the occurrence of the inherited syndrome throughout as many as four or five generations, such instances being fairly common (Adair-Dighton, Berneaud, Bronson, Burrows, Cockayne, Conlon, Knapp, Paul, Peters, Spurway, Stephenson-Harman, Stobie [8]). In some of the affected subjects all three symptoms of the syndrome were present; in others, only one or two. Particularly in the older literature,

6. Boyd, W.: A Text-Book of Pathology, ed. 2, Philadelphia, Lea & Febiger, 1934.

7. Gutzeit, R.: Klin. Monatsbl. f. Augenh. **68**:771, 1922.

8. Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1932.

the presence of otosclerosis is often not reported. Only in comparatively recent years was attention called to the combination of deafness with fragilitas ossium and blue scleras. Furthermore, the deafness, which in these cases is hereditary, develops often after the age of 20 and is due to secondary changes suffered as sequelae of the multiple fractures, or, owing to a possibly lessened general resistance, life is usually terminated by an intercurrent disease, such as pneumonia, before the deafness manifests itself. The close association of the two cardinal symptoms, i. e., idiopathic spontaneous fractures and blue scleras, was recognized definitely by Eddowes.

The blue color of the scleras resembles that met with in staphyloma sclerae. The intensity of the hue varies with, and is more or less accentuated in, individual cases. As a result different authors have used varying descriptive terms, such as blue-gray, sky blue, blue with a yellowish tinge, china blue, brilliant blue and intensive blue. The discoloration is usually diffuse, beginning at the limbus and continuing backward. In one of Hirschmann's cases [9] the patient had a ring of normal color about 2 or 3 mm. wide around the limbus; then it shaded into intensive blue. Among our patients one showed a marked deep blue ring-shaped area surrounding the limbus, which gradually changed into a less intensive blue. In cases of intensive blue scleras the contrast to the normal coloring is extremely marked. In cases of mild involvement, however, differentiation from the normal may be difficult, since many new-born children and even adults, especially those of dark-pigmented races, occasionally show a slight but definite bluish color of the sclera. Abnormal translucency (Gutzeit, Blatt, Freytag) is not an absolute criterion. There are borderline instances in which variation of a pathologic blue color from the normal is so insignificant as to cause some hesitation in making a diagnosis of blue scleras.

The underlying pathologic condition is either abnormal thinness of the sclerotica or lack of deposits of calcium within the outer coat of the eye. Buchanan reported that in this condition he noted that the thickness of the cornea was three fourths of the normal, and that of the sclera, one third of the normal, with absence of Bowman's membrane and diminution of the fibers, observations which were recently confirmed by Casanovas.[10] These observations were contradicted by the results of anatomic examination in a case of blue scleras reported by Bronson and by the observations made by Wirth and Vogt with the aid of the slit lamp. It therefore can be readily assumed that the blue coloration must be only the result of a changed transparency to light, no anatomic change being microscopically discernible.

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9. Hirschmann, J.: Ztschr. f. klin. Med. **126**:718, 1934.
10. Casanovas: Zentralbl. f. d. ges. Ophth. **31**:580, 1934.

With blue scleras and spontaneous fractures there are frequently further complications of the bones, such as prominent frontal and occipital bones (Boyd, Blatt, Bronson, Stroble), frequent luxations of the interphalangeal joints, especially of the thumb (Behr, Cockayne, Conlon, Crocco, van der Hoeve), and kyphosis, lordosis and scoliosis. The deafness may be attributed to otosclerosis due to changes in the petrous bone and calcification in the labyrinth (Stenvers, Stobie), combined with partial stapes ankylosis (Bigler, Gimplinger). Disturbance of the calcium metabolism, resulting in late dentition (Crocco, Paal), and brittleness of the teeth (Paal, Stobie, Terrien) may also be associated with the condition. Other complications may be present, such as syndactylism (van der Hoeve), mongoloid idiocy (Bolton), vitium cordis, palatum fissum (Voorhoeve), conical cornea (Behr, Urbanek) and zonular cataract (Blegvad and Haxthausen)

HYPERPARATHYROIDISM

In order to place the syndrome of blue scleras within or outside the domain of endocrine disturbance, especially hyperparathyroidism, it is necessary to review the known facts concerning parathyroid disorders. This knowledge has been extensively modified in the last decade. As in the development of many other medical problems, orientation is rather difficult because of contradictory observations and unjustified conclusions. It would lead far afield to give all the details concerning parathyroid disorders. Therefore, only those essential to the understanding of the subject are here discussed.

The pioneer work in this field was done by MacCallum and Voegtlin, who discovered that in parathyroidectomized dogs the calcium content of the blood serum is reduced. This was later strongly confirmed by further research. Since the isolation of the active principle of the parathyroid gland by Hanson (1924) and Collip (1925) it has been repeatedly demonstrated that the injection of parathyroid extract produces marked elevation of the blood calcium level. Brehme and György found, in addition, that parathyroid extract lowers the blood phosphate content and causes a shift in the p_H of the blood toward the acid side without disturbing the carbon dioxide-combining power of the blood. Reiss and Aub, working independently, found lowering of the blood phosphorus level due to injections of parathyroid extract. The various authors unanimously agreed that such injections in normal persons result in a rise of the blood calcium content above the physiologic level, accompanied by an increased calcium content of the urine (even to six times the normal). The increase in calcium salts they considered to be due to their mobilization from the bones, which store compounds of this element just as the liver stores glycogen. Thus the conclusion

is obvious that the physiologic function of the parathyroid glands is the exact regulation of the calcium metabolism, i. e., control of the rate at which the calcium is moved from its deposits, namely, the bones, into the blood stream and tissues and, finally, into the urine. The amount of calcium present in the tissues is responsible for their neuromuscular irritability, which varies inversely with the calcium content (hypotonia of the muscles is associated with hyperparathyroidism and hypertonia of the muscles with tetany). Pathologic disturbance of the parathyroid glands may lead to either hyperparathyroidism or hypoparathyroidism, the former resulting in the presence of a negative calcium balance and generalized osteitis fibrosa cystica, and the latter, in tetany accompanied by a positive calcium balance (Goerner and Samuelsen [11]) with characteristic hyperirritability, known clinically as the Chvostek and Trousseau signs.

The increased activity of the parathyroid glands, as has already been mentioned, results in hypercalcemia of the blood arising from excessive loss of calcium from the body by way of the kidneys, the level of calcium in the blood, however, being only a rough indication. The exact evaluation of the calcium metabolism can be obtained solely by careful study of the calcium balance. Hunter and Turnbull [12] strongly advocated determination of the calcium balance and stressed the comparison that "just as the level of a river does not indicate the direction in which it flows, so the levels of the calcium in the blood give us, in themselves, no indication as to whether the calcium is flowing into the excretory channels or into the tissues."

Method of Estimating the Calcium Balance.—In order to establish the calcium balance the patient has to be kept on a fixed diet of sufficient caloric value with a low calcium content (100 mg. a day). The intake of calcium must be closely observed, and only distilled water should be used both for drinking purposes and in the preparation of meals. In addition, the intake of sodium chloride has to be controlled. Sufficient sodium bicarbonate should be given by mouth to make the reaction of the urine neutral, since with an acid medium the figures for calcium are necessarily higher. As the calcium is usually eliminated through the kidneys, the calcium content of the urine has more significance than that of the feces. Only in pathologic conditions, such as calcification of the kidneys (which may be associated with deposits of calcium in other organs, such as the lungs and spleen, in hyperparathyroidism), is there no increased excretion of calcium in the urine, for, owing to the inability of the kidneys to eliminate the extra load, it will appear in the

11. Goerner, A., and Samuelsen, G.: Calcium Metabolism in Idiopathic Hypoparathyroidism, J. A. M. A. **102**:1001 (March 31) 1934.

12. Hunter, D., and Turnbull, H. M.: Brit. J. Surg. **19**:203, 1931.

feces. In such cases the quantity of calcium in the feces must be determined. The most reliable method seems to be either that of Kramer and Tisdall or the modification of this by Clark and Collip.

The normal content of calcium in the blood is between 9 and 11 mg. per hundred cubic centimeters. Increase of the blood calcium level with simultaneous increase of the excretion of calcium, when the intake is controlled, is called a negative calcium balance and is characteristic of hyperparathyroidism. The injection of an extract containing the active principle of the parathyroid gland in normal persons and in animals produces a similarly high calcium level in the blood serum and an increase of excretion of calcium in the urine, not infrequently reaching a figure six times the normal level.

Rôle of the Phosphorus Content.—In hyperparathyroidism the high level of the calcium in the blood and the increased excretion of calcium in the urine are associated with a low level of phosphorus in the serum. This glandular hyperactivity is almost unique in producing these simultaneous changes. Numerous conditions may cause an increase in the calcium level of the blood (multiple myeloma and metastatic malignant growth) or a low phosphorus content (rickets and osteomalacia), but the singular combination of an increased calcium content and a low phosphorus content is pathognomonic of hyperactivity of the parathyroid glands. According to Aub and Bauer, a serum calcium level above 11 mg. per hundred cubic centimeters and a phosphorus level below 3.5 mg. per hundred cubic centimeters should be regarded with suspicion, especially if repeatedly noted.

Method of Determining the Phosphorus Content.—Fresh blood should be used in making the determination of the phosphorus content, for if the blood has been allowed to stand the phosphorus content shows variation. When the corpuscles have been laked the liberated organic esters are rapidly hydrolyzed by the plasma phosphatase, and the concentration of the inorganic phosphates rises rapidly. Therefore, the blood should be immediately oxalated, and hemolysis should not be permitted. With such precautions the experimental error in the method of Briggs (used by Hunter and Turnbull) should not exceed 2 per cent, a phosphorus content of between 2.5 and 3.5 mg. per hundred cubic centimeters being considered normal. Aub and Bauer used the method of Frike and Subbarow and regarded any level below 3.5 mg. as indicative of hypophosphaturia.

In seventy-three of seventy-eight cases reported by various workers, all of which were unquestionable instances of hyperparathyroidism, determinations consistently showed a serum calcium content above 11.5 mg., and in 59 cases there was a constant level above 12 mg.

Phosphatase.—The hypercalcemia and the associated decrease in the inorganic phosphorus content are accompanied by increased activity of the blood phosphatase. The latter is an enzyme that plays a specific rôle in the deposition and maintenance of the calcium and phosphorus compounds in the tissues. An increased concentration of the enzyme in the plasma is found in several diseases in which there is abnormal destruction or formation of bone tissue, such as Paget's disease, mild and healing rickets, certain types of osteomalacia and obstructive jaundice. Very high values for phosphatase are encountered particularly in active rickets and generalized Paget's disease. However, only hyperparathyroidism is known as a clinical entity in which a markedly increased phosphatase content in the blood serum (from 12 to 25 Bodansky units) is combined with a negative calcium and a negative phosphorus balance. The phosphatase level of from 2 to 4 units is considered normal and is probably an index of the osteoblastic activity. It is elevated in hyperparathyroidism in direct proportion to the extent of the osseous disorder and is independent of the degree of parathyroid disturbance. Accordingly, osseous disturbances of various origins may result in increase of the phosphatase content, but in such cases there will be no negative calcium and phosphorus balances. Observation of persons after operation for parathyroid tumors shows a dramatic change in the calcium and the phosphorus metabolism, but, on the other hand, the phosphatase level, after a period of months, only gradually returns to the normal.

Interpretation of Findings.—The variation between what is pathologic and what is physiologic, especially at the beginning of any disease, may be so slight that differentiation is not sharp. Hyperactivity of the parathyroid glands may be present without an appreciable increase in phosphatase activity in cases in which the alterations in the osseous substance are not yet far advanced or in those instances in which shifting of the calcium and phosphorus levels can be detected only after repeated examination. However, the laboratory findings of an increased blood calcium level and a decreased blood phosphorus level, associated with increased excretion of calcium and phosphorus in the urine and an elevated phosphatase level of the blood, should strongly suggest hyperactivity of the parathyroid glands.

Osseous Condition.—It may be definitely stated that the characteristic laboratory findings demonstrate a profound disturbance in the calcium metabolism, viz., the inability of the bones to store and retain calcium, which is the most outstanding symptom of parathyroid hyperactivity and must result in generalized decalcification of the skeleton. The typical skeletal changes were first described by von Recklinghausen [13] as osteitis fibrosa cystica, and thirteen years later Askanazy

13. von Recklinghausen, F. D., in Festschrift für Rudolf Virchow, Berlin, G. Reimer, 1891.

first pointed out the relationship of this condition of the bones to a tumor of the parathyroid gland in his case. Since then further observation, study and isolation of the parathyroid hormone have supplemented the knowledge of the physiology of the parathyroids and proved that Recklinghausen's disease is a clinical manifestation of parathyroid hyperactivity and that an identical osseous disorder can be produced experimentally in animals by injections of parathyroid hormone.

Generalized osteitis fibrosa consists of widespread resorption affecting all the bones, resulting in osteoporosis, cyst formation and benign giant cell tumors the cells of which are similar to the osteoclasts normally present in Howship's lacunae. The excessive stimulation of osteoclastic resorption is accompanied by new formation of fibrous tissue occupying the dilated haversian canals, i. e., marked fibrosis of the marrow (in the recent German literature osteitis fibrosa cystica is referred to as osteodystrophia fibrosa). Some of the connective tissue may become converted into osteoid tissue, formation of new bone taking place with rows of osteoblasts, but resorption greatly preponderates and outstrips ossification and apposition, while osteoclasts and giant cell tumors grow to larger proportions. The presence of osteoclastoma or giant cell tumors is a characteristic feature of the picture of hyperparathyroidism. The newly formed connective tissue is poorly vascularized, and as a result degeneration, softening and cyst formation frequently occur. The osteoclastomas and cyst formation are important factors in differentiating osteitis fibrosa cystica from generalized osteoporosis, and one must bear this in mind in the discussion of the latter disease.

A negative calcium balance and generalized osteitis fibrosa cystica are the distinguishing features of parathyroid hyperactivity, which may be caused by a malignant growth or by so-called adenoma of these glands. Other clinical features may be found, among which Bauer [14] enumerated polydipsia, polyuria, general malaise, constipation, anorexia, loss of weight, vague muscular and articular pains, tenderness of the bones, frequent fractures, decreased excitability of the nerves, skeletal shortening, kyphosis, osseous tumors, stones in the kidney and ureters and, frequently, anemia with leukopenia. The osseous changes consist of generalized decalcification, cyst formation and tumors, as was previously mentioned.

The disease entity which was primarily known as Recklinghausen's disease and later known as osteitis fibrosa cystica and hyperparathyroidism was first recognized by Mandell in Vienna and by DuBois in America. At the present time the literature contains reports of more than a hundred cases in which the clinical and the laboratory diagnosis

14. Bauer, W. J.: J. Bone & Joint Surg. **15**:135, 1933.

have been confirmed by operation. The pathologic observations and ensuing postoperative recovery jointly clarified the picture. In the hundred cases in which an operation was performed, about one fifth of the patients were under observation and operated on at the Massachusetts General Hospital. The work of Churchill, Albright and Bauer and their co-workers,[15] therefore, should have much weight in a discussion of the subject. Churchill emphatically warned against the danger of overstepping in this new field of endocrine disturbance and advocated as next in importance to the study of various clinical symptoms the careful consideration of the calcium metabolism and the roentgenographic observations on the bones.

This author also took an emphatic stand against the removal of one or more of the parathyroid glands (including interference with their blood supply) for several conditions other than hyperparathyroidism and called the evidence supporting such recommendations highly inconclusive. He referred particularly to the work of Oppel [16] and of Ssamarin, who performed parathyroidectomy in about seventy cases of ankylosing polyarthritis. Leriche, and Ballin, in this country, resorted to the same treatment. The latter was even more radical, advocating parathyroidectomy for Paget's disease as well as for ankylosing polyarthritis. This is of great importance in consideration of the types of cases in which there are blue scleras and osteochondritic changes. The latter condition is usually regarded as a form of arrested embryologic development and therefore is excluded from the endocrine type of disorders. Ballin was carried away by the conception that hyperparathyroidism, either fetal or acquired early in life, may be the responsible factor in the mechanism involved in these cases and advised reinvestigation of all juvenile types of osseous disturbances, particularly fragilitas ossium and achondroplasia.

DIAGNOSIS OF PARATHYROID HYPERACTIVITY

There is a wide difference between the opinions of Churchill and Ballin. The former insisted that the classic picture must be present before parathyroidectomy is indicated; the latter drew criteria for surgical intervention much wider in scope. A pertinent question naturally

15. (*a*) Albright, F.; Aub, J. C., and Bauer, W.: Hyperparathyriodism: A Common and Polymorphic Condition as Illustrated by Seventeen Proved Cases from One Clinic, J. A. M. A. **102**:1276 (April 21) 1934. (*b*) Churchill, E. D., and Cope, O.: Surg., Gynec. & Obst. **58**:255, 1934. (*c*) Albright, F.; Bloomberg, E.; Castleman, B., and Churchill, E. D.: Hyperparathyroidism Due to Diffuse Hyperplasia of All Parathyroid Glands Rather Than Adenoma of One, Arch. Int. Med. **54**:315 (Sept.) 1934.

16. Oppel, W. A.: Endokrinologie **6**:11, 1930.

arises, viz.: What factors led to such opposing views of the indications for operative intervention on the parathyroid glands?

Churchill and his co-workers based their diagnosis of parathyroid disturbances mainly on the evidence of a negative calcium balance combined with the characteristic roentgenographic findings of the skeleton. In their evaluation of symptoms the associated clinical manifestations, which were enumerated by Bauer, were considered of secondary importance. Certain other clinical features may be either present or absent during the course of the disease in different cases. These are calculus formation in the kidneys, muscular atony and changes in the terminal joints of the fingers (which, according to Keynes and Taylor, are very short and square, with correspondingly stubby and broad nails). The slightly bulbous finger ends should not be confused with the clubbing seen in pulmonary osteo-arthropathy. The most reliable diagnostic evidence of osteitis fibrosa cystica is obtained by roentgenography.

Decalcification of the bones may occur in various diseases and has been observed in association with disuse, arthritis, fractures, starvation, rickets, osteomalacia and celiac disease associated with rickets (all of which are apparently dependent on vitamin D deficiency), but these forms of decalcification are entirely different from that in osteitis fibrosa cystica and do not belong in the same group. Camp [17] insisted that osteoporosis alone is not sufficient evidence on which to base a roentgenographic diagnosis of parathyroid disorder and described the osteoporosis in osteitis fibrosa cystica as uniform and granular, stressing the fact that its peculiar form of mottled atrophy is distinct from the ordinary type seen in osteoporosis associated with acute and chronic disease of the bones and neurotrophic conditions.

In contradiction to definite laboratory findings, Oppel, Leriche, and Ballin [18] failed to establish an accurate calcium balance and were satisfied with an occasional determination of the calcium content of the blood serum. Ballin, following Oppel, emphasized the importance of muscular hypotonia associated with parathyroid disturbances and pointed out four distinct diagnostic signs: (1) the determination of the number of milliamperes necessary to provoke muscular contraction; (2) chronaxia, which was introduced by Lepique, as an electric time coefficient of neuromuscular excitability (according to Bourguignon, increase of chronaxia in hyperthyroidism is more reliable than the serum calcium content); (3) demonstration in a motion picture of the slow or weak action of muscles, and, finally, (4) shortening of the RT wave in the electrocardiogram, due to myocardial weakness. Ballin attached great signifi-

17. Camp, J. D.: Osseous Changes in Hyperparathyroidism, J. A. M. A. **99**:1913 (Dec. 3) 1932.

18. Ballin, M.: J. Bone & Joint Surg. **15**:120, 1933; Ann. Surg. **96**:649, 1932; **98**:868, 1933.

cance to muscular hypotonia in establishing the diagnosis and included in the syndrome of hyperparathyroidism clinical entities varying from typical generalized osteitis fibrosa cystica, Paget's disease in his opinion merging into it without any striking differences. Such an assertion does not seem to be valid, considering all the known evidence that the two diseases are distinct entities, and stands in contradiction of the findings of Schmorl, who in a careful investigation of one hundred and ninety cases of Paget's disease failed to encounter symptoms of hyperactivity of the parathyroid glands.

Critical analysis of the literature leads us to the single conclusion that parathyroid hyperactivity should be suspected only if there are a negative calcium and a negative phosphorus balance and an increased phosphatase content, associated with roentgenographic findings in the skeleton characteristic of osteitis fibrosa cystica. In similar conditions the clinical picture may show various features, but the underlying disorder must give evidence of the same cardinal characteristics. The osteoporosis encountered in different conditions, especially that associated with blue scleras, is an entirely different pathologic entity, not based on disturbances of the parathyroid glands.

CHANGES IN THE BONE ASSOCIATED WITH BLUE SCLERAS

The skeletal changes in connection with blue scleras are described as osteopsathyrosis or osteogenesis imperfecta. Many authors are of the opinion that these are merely two aspects of the same condition, both showing evidence of deficient osteoblastic function. Looser, therefore, made the suggestion that "the essential unity of the two conditions should be recognized by giving osteopsathyrosis the name of osteogenesis imperfecta tarda." Bauer took an identical stand, affirming the existence of embryologic defectiveness.

According to Bauer, osteogenesis imperfecta is a condition involving the connective tissue as a whole, manifesting itself as a dysfunction of all the matrix-forming cells. While all tissues of mesenchymal origin are affected, the more highly organized ones (bone, cartilage and tooth pulp) are chiefly concerned in the disease process. That the cell formation is not necessarily deficient is exemplified by the case reported by Buday, in which osteoblasts were present in great numbers and exhibited normal structure but their matrix-forming function was impaired. Fahr and Krauss failed to confirm Bauer's theory of dysfunction of the matrix-forming cells. Fahr reported excessive development of the thyroid and thymus (which are derived from entoderm) and of the pituitary and adrenal medulla (an ectodermal derivative), but his objections were based mainly on a singular unconfirmed case.

Various observers have described changes in other tissues of mesodermal origin, such as muscular hypotonia and an excess of lymphocytes in the blood (Frontali, Macciotta). Other authors, differing with Bauer, pointed out involvement of tissue of epiblastic origin (enamel, the auditory nerve). As a result mesenchymal deficiency is not generally accepted as a true and sufficient explanation of the osseous changes associated with blue scleras. Many of the recent authors agreed that the lack of osteoblasts (due to dysfunction, according to Bauer) is the principal characteristic in the histologic picture of osteogenesis imperfecta in the syndrome of blue scleras.

The question then arises as to whether the skeletal defectiveness present might be traceable to an original malformation, or vitium primae formationis (Kaufman, Dicherle, Sumita), or to disturbances influencing the bone-forming tissues only secondarily. The most exhaustive microscopic study was made by Weber,[19] in the case of a girl 2 days old with early and recent fractures of the long bones and ribs (with no mention of blue scleras). This case demonstrated that osteogenesis imperfecta is a generalized disorder affecting similarly the entire bone-building system and that its origin is traceable to an early stage of intra-uterine life.

According to Weber, the picture of osteogenesis imperfecta is one of arrested development with active and passive manifestations. The passive manifestation of arrested development is the lack of the highest unit, the osteon. Either no osteoblasts are present, or they are unable to perform their function—the production of shell bone. This arrestment in the destined development is primary, so that the formation of lamellar bone never occurs.

The active manifestation is encountered in the bone tissue actually formed, the entire skeleton consisting of imperfect fiber bone. The latter is ontogenetically and phylogenetically the oldest type of bone tissue, forming the nucleus for subsequent deposition of the shell bone. Thus in osteogenesis imperfecta the formation of bone tissue is arrested in the stage of nucleus formation.

Weber described the pathologic changes of osteogenesis imperfecta as a relative shift to the left. If an attempt were made to present the separate stages of bone formation graphically, the highest one on the extreme right would be lacking. In the middle stages bone formation would be seemingly increased (whereas it is only relatively increased); that is, the fiber bone is formed in normal quantity but is not resorbed by the osteoblasts, owing to their absence. In the lowest stage bone formation would be less than normal. An important sign is the inferior

19. Weber, M.: Osteogenesis Imperfecta Congenita, Arch. Path. 9:984 (May) 1930.

quality of the intercellular substance in the fiber bone. Since this pathologic fiber bone has its origin in embryonal tissue, the conclusion is confirmed that this condition is a congenital malformation.

Weber agreed with Kaufman and Bauer in assuming a germinal and not a secondary disturbance, for in osteogenesis imperfecta the osteoblasts and giant cells either are lacking or are failing in function. Both these cellular conditions originate in the endothelial cells of the blood vessels, and thus the latter are responsible for the changes in the bone formation.

The occurrence of severe arteriosclerosis in some cases of osteogenesis imperfecta also points to vascular involvement. The juvenile type of arteriosclerosis indicates a disturbance in the composition of intercellular substances similar to that in the pathologic formation of fiber bone. Thus the changes are traceable as far back as mesenchymal development, and the analysis and minute observation made by Bauer on mesenchymal dysfunction are confirmed.

METABOLIC CHANGES IN CASES OF BLUE SCLERAS

The pathologic changes in osteogenesis imperfecta and osteitis fibrosa cystica are, therefore, well characterized and well defined. It is easily perceived that the underlying pathologic picture of each is absolutely different. Evidently, the changes in the calcium and the phosphorus metabolism in osteogenesis imperfecta associated with blue scleras should vary from the usual negative balance in cases of osteitis fibrosa cystica, which is the clinical manifestation of osseous changes in hyperactivity of the parathyroid glands.

Bookman,[20] without mentioning blue scleras, reported that in cases of active osteogenesis imperfecta the retention of calcium is somewhat or decidedly below normal. Similar statements relative to a retention of calcium lower than normal were made by Schabad.[21] In a child 7 months old with this osseous condition Schwarz and Bass [22] found a positive calcium balance, a retention of calcium somewhat below normal and a practically normal phosphorus metabolism; no reference to blue scleras was made. According to af Klercker,[23] in this condition the retention of calcium is normal but at its lower range, while Flagstad, Zanger and Leven [24] cited a normal concentration of both calcium and phosphorus.

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20. Bookman, A.: The Metabolism in Osteogenesis Imperfecta with Special Reference to Calcium, Am. J. Dis. Child. **7**:436 (June) 1914.

21. Schabad, I. A.: Ztschr. f. Kinderh. **11**:230, 1914.

22. Schwarz, H., and Bass, M. H.: Osteogenesis Imperfecta: Report of a Case with the Study of Its Metabolism, Am. J. Dis. Child. **5**:131 (Feb.) 1913.

23. af Klercker, K. O.: Monatschr. f. Kinderh. **25**:338, 1923.

24. Flagstad, A. E.; Zanger, E., and Leven, L.: Minnesota Med. **7**:800, 1924.

In Hunter's [25] typical case a boy aged 17, with an irrelevant family history, had blue scleras, associated with multiple fractures (more than thirty), which began at the age of 3 weeks. Hunter considered the negative calcium balance as due to the low intake, for in the control case the determination was identical, and he therefore concluded there was no increased loss of calcium. Tauber [26] observed two sisters, the older of whom showed marked osteogenesis imperfecta while the younger showed only traces of this disease. In both cases the negative calcium balance was improved by the use of different therapeutic measures. Sindler's [27] patient, a 20 day old child, showed a serum calcium content of 7.8 mg. per hundred cubic centimeters and a normal calcium metabolism. Two patients examined by Holler [28] had blue scleras and multiple fractures and showed a blood calcium content of 0.015 per cent, estimated by the Jansen method—a slight increase above the normal (0.01 per cent). Wyatt and McEachern's [29] patient, an infant 4½ weeks old, had a serum calcium content of 10.2 mg. and a phosphorus content of 6.5 mg. per hundred cubic centimeters, the calcium and phosphorus contents of bone ash and their ratio being normal. In Kay's [30] opinion, there is a tendency to higher values than normal for calcium and phosphorus in fragilitas ossium but no constant increase of phosphatase. The average value for phosphatase in four cases was 0.5 unit, the normal for the age group being 0.22 unit. In some cases of osteitis fibrosa generalisata, osteitis deformans, osteomalacia and rickets the value for phosphatase rises above 3 units, which is more than twenty times the normal average.

Two patients with osteogenesis imperfecta were observed by Hassman and Verdino.[31] The first showed higher values than normal for calcium and normal values for phosphorus in the whole blood and an increased amount of phosphorus in the serum. The second patient showed a similar condition, but in this case the serum phosphorus content was normal. In their observation of the disease Bodansky and Jaffe [32] found a normal average for the phosphatase content in the blood serum—about 2.7 and 8 units per hundred cubic centimeters in adults and children, respectively—but a greatly increased value in polyostotic Paget's disease (fifty times greater than normal), in rickets (twenty

25. Hunter, D.: Lancet **1**:9, 1927.
26. Tauber, M.: Monatschr. f. Kinderh. **36**:12, 1927.
27. Sindler, A.: Ztschr. f. Kinderh. **42**:85, 1926.
28. Holler: Verhandl. d. deutsch, orthop. Gesellsch. **20**:273, 1925.
29. Wyatt, T. C., and McEachern, T. H.: Congenital Bone Dysplasia (Osteogenesis Imperfecta) Associated with Lesions of the Parathyroid Glands, Am. J. Dis. Child. **43**:403 (Feb.) 1932.
30. Kay, H. D.: J. Biol. Chem. **89**:249, 1930.
31. Hassman, K., and Verdino, A.: Jahrb. f. Kinderh. **140**:1, 1933.
32. Bodansky, A., and Jaffe, H. L.: J. Biol. Chem. **105**:11, 1934.

times greater than normal) and in hyperthyroidism (ten times greater than normal), a lower increase in generalized osteoporosis and osteomalacia and a similar but inconstant content in fragilitas ossium.

In four cases of osteogenesis imperfecta Hansen [33] observed values for phosphatase almost identical with those in the control cases of subjects of the same age group. Aub and Farquharson [34] reported one case of fragilitas ossium not associated with blue scleras, in which repeated fractures occurred from the slightest trauma, the roentgenograms showing osteomalacia. The excretion of calcium with a low calcium diet was normal, although the urinary output of calcium is probably a little higher than normal in most children of the patient's age. The calcium content of the blood serum was normal; the phosphorus content was slightly above the usual level for children.

Takahashi reported four cases of blue scleras in which there was an increased urinary output of calcium as compared to the normal output of calcium in three other cases. In his study the calcium balance was not established. The calcium level in the blood was examined in only one of the four cases, and a calcium content of 5.6 mg. per hundred cubic centimeters was reported, in contrast to the normal calcium levels of two controls (11.43 mg. and 9.97 mg.). This hypocalcemia led him to conclude that there was hypo-activity of the parathyroid glands, an assertion which was generally adopted and which was quoted many times in the literature. The validity of such an assertion must not be too hastily accepted, particularly since many other authors, using the same technic, could not establish such a low calcium level in the blood. Paal,[35] in his first case, found 9.066 mg.; in the second case, 9.99 and 9.73 mg., and in the third, 12.53 and 12.8 mg., the latter values being somewhat pathologically increased. The author remarked that in the last case the roentgen ray translucency was not typical, while it was typical in the two former cases. Stevenson and Cuthbertson,[36] examining the calcium and phosphorus levels in four cases in which the intake of calcium was controlled, found these to be within the normal limits in all instances. Examination of the urine and feces for the retention of calcium oxide (CaO) and phosphorus pentoxide (P_2O_5) showed a diminished amount in two cases (in one the retention of magnesium was also much diminished) and an almost normal content in the third case. In the fourth case the metabolic investigation was made eleven weeks after a serious accident resulting in multiple fractures and revealed a decidedly diminished retention. At that time there was still

33. Hansen, A. E.: Proc. Soc. Exper. Biol. & Med. **31**:1023, 1934.

34. Aub, J. C., and Farquharson, R. F.: J. Clin. Investigation **11**:235, 1932.

35. Paal, H.: Klin. Wchnschr. **8**:1304, 1929.

36. Stevenson, G. H., and Cuthbertson, D. P.: Lancet **2**:782, 1931.

absence of callus formation. A few weeks later, when the callus had formed, the retention of calcium oxide and phosphorus pentoxide became less marked.

In a typical instance of osteogenesis imperfecta and blue scleras, in which ten days after birth a fracture of the humerus was present and during the ensuing ten years numerous spontaneous fractures were sustained, the blood calcium level, as determined repeatedly by Gyllenswärd,[37] was 8.6, 9.6 and 9.9 mg. per hundred cubic centimeters. Ottley [38] reported 10.2 mg. of calcium in the blood serum. Hirschman in his first case found 13.06 mg. and in the second 16 mg. These rather high levels, in his opinion, did not indicate any endocrine involvement. Unfortunately, Hirschman failed to establish the calcium balance in his cases. As most of these authors omitted the value for phosphatase in all these metabolic determinations, their figures are not enlightening.

Kleinberg [39] recently reported two cases of osteogenesis imperfecta, with accurate laboratory findings. The first patient, a 2 month old child, who had exhibited fractures at birth, had a blood calcium content of 10.3 mg., a blood phosphorus content of 5.6 mg. and a phosphatase content of 15.6 units. The second patient, a 2 year old child with deep blue scleras (and proof of blue scleras in the antecedents) showed normal values for calcium, phosphorus and phosphatase. The blood calcium content amounted to 10.5 mg. in the first case reported by Harnett [40] and to 10 mg. in the second case (in which the mother, brother and sister also exhibited blue scleras).

It is evident that there is a lack of uniformity in the laboratory determinations and findings in the cases of blue scleras so far reported. In order to elaborate on the laboratory data in cases of osteogenesis imperfecta we report two cases which we had an opportunity to observe in the wards of the Beth Israel Hospital. Not only were the examinations of the serum calcium accurately made, but the calcium metabolism was carefully studied. To add to the latter study, the values for phosphorus and phosphatase were also determined, and roentgenographic examinations were made.

REPORT OF CASES

CASE 1.—*History.*—A white boy, 12 years old, with markedly blue scleras since birth complained chiefly of inability to walk and the past occurrence of repeated fractures on sustaining trivial injury. The fractures were as follows: (1) fracture of the right clavicle, which was sustained when he was being lifted out of a carriage at the age of 5 months; (2) fracture of the right clavicle again

37. Gyllenswärd, C.: Acta pædiat. **13**:111, 1932.

38. Ottley, C. M.: Arch. Dis. Childhood **7**:137, 1934.

39. Kleinberg, S.: J. Bone & Joint Surg. **16**:953, 1934.

40. Harnett, W. L.: Brit. J. Surg. **22**:269, 1934.

when he was 2 years old; (3) fracture of the left wrist at 7 years of age; (4) fracture of the left wrist a second time, at 8 years of age, and (5) fracture of the left femur at 10 years of age.

All the fractures healed normally. The patient was able to walk, but with difficulty, until the left femur was fractured, when the use of crutches became necessary. In addition he had congenital clubfoot.

Family History.—No other members of the family had any osseous dyscrasias.

Physical Examination.—The boy was well developed and obese, measuring 5 feet and 2 inches (157.5 cm.) in height and weighing 125 pounds (56.7 Kg.). The blood pressure was 120 systolic and 88 diastolic.

Head: The occipitofrontal circumference was 22 inches (55.8 cm.). There was bitemporal bulging.

Eyes: Both scleras were deep china blue, especially in a ring about 0.5 cm. around the limbus. Toward the equator the color become less intensive. The pupils were equal and regular and reacted well to light and in accommodation. The fundi were normal. The conjunctivae showed a good hemic component.

Nose, Mouth and Teeth: These were normal.

Chest: On inspiration the circumference measured 34 inches (86.4 cm.); on expiration it measured 33½ inches (85.2 cm.). Both breasts were large and obese; no fluid could be expressed.

Heart: This organ was not enlarged. The sounds were regular and of good quality. The pulmonic second sound was greater than the aortic second sound.

Extremities: These were obese and hypotonic. The wrists were double-jointed. There was a definite weakness in the grasp of the hands, especially of the left hand, and a weakness of the extensor muscles of the thighs. There was a tendency to bilateral foot drop, although the feet could be flexed. The right foot was held in an abductor position from the midline, and the left foot was held in adduction (toward the midline). There was some tendency toward knock knee (genu valgum). The patient could not raise his legs (to put on stockings) without the support of his arms.

The fingers were long, thin and tapering, with lateral curvature of the nails and a slight cyanotic hue. The little finger of the left hand was deformed, owing to a lateral curvature of the first two phalanges (caused by an old fracture).

Measurements: Both the left and the right biceps measured 10 inches (25.4 cm.). The distance from the acromion process of the clavicle to the inner condyle of the ulna was 21 inches (53.3 cm.) on both the right and the left. The midportion of the thigh measured 17 inches (43.2 cm.) on both the right and the left. The lower third of both tibias measured 11½ inches (29.2 cm.). The distance from the left anterior-superior spine to the interior malleolus on the right was 33¾ inches (85.7 cm.) and to that on the left, 35 inches (88.9 cm.). A scar 3 inches (7.6 cm.) long was present on the inner aspect of the right foot and above the right heel, and another scar, 5½ inches (13.9 cm.) long, was seen on the lower half (inner aspect) of the right leg. The scar formation was due to previously performed bone graft from the right tibia into the right os naviculare. No callus formation was present.

There were bowing and thickening of the distal third of the left radius anteriorly, and a small dorsal deformity was felt in the wrist of the left hand.

Reflexes: The knee jerks were present and equal. The biceps reflexes were present but sluggish. No pathologic reflexes were elicited.

Posture: Slight scoliosis (an old spinal curvature corrected by a brace and the application of a cast) was present.

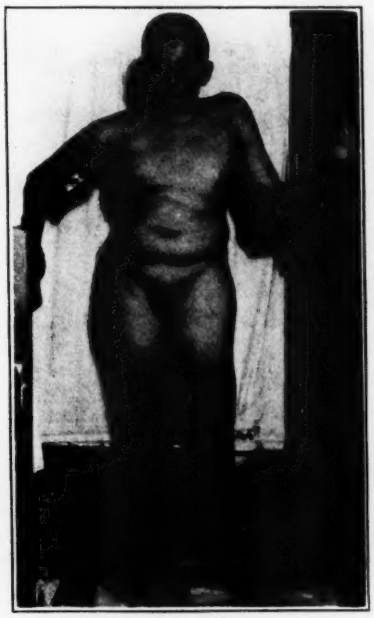

Fig. 1 (case 1).—Clinical appearance of patient. Owing to the multiple fractures suffered at various periods, he has to be supported.

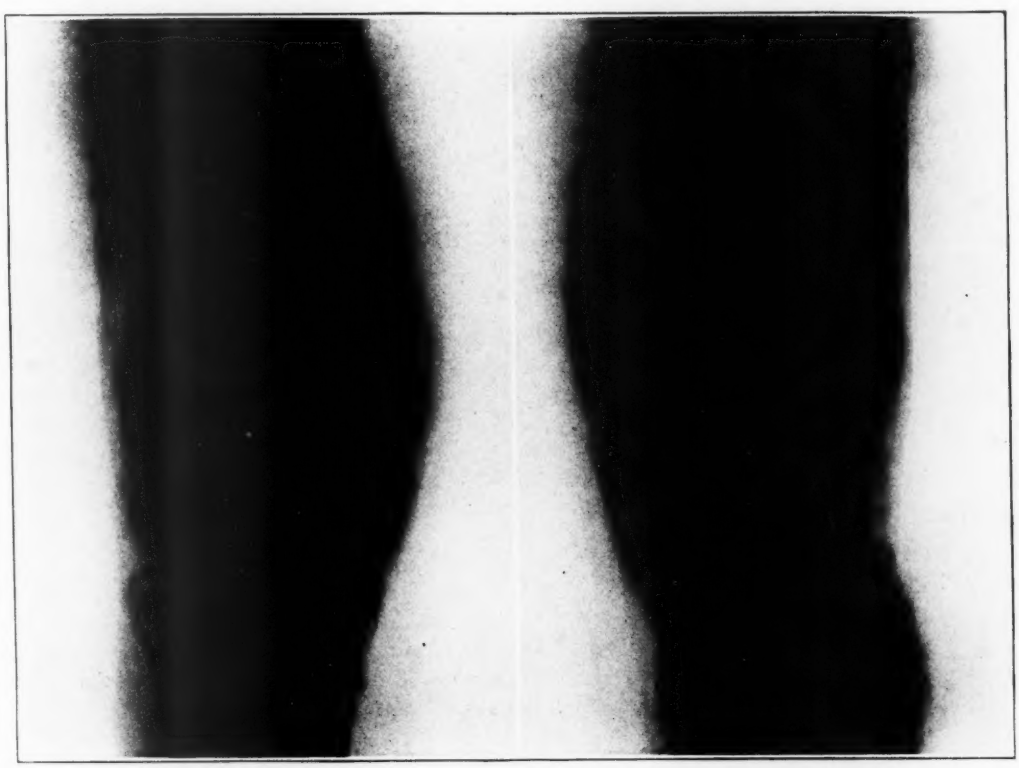

Fig. 2 (case 1).—Roentgenogram of the knees of patient. The osteoporosis is extremely marked, and the decalcification of all the bones is pronounced. The shaft of the left tibia shows evidence of old fractures.

Joints: These were lax, indicating hypotonicity of the ligaments and tendons. There was hyperextension of the fingers at the interphalangeal, metacarpophalangeal and wrist joints. When the hand was held outward it fell as in wrist drop. At the elbows there were hyperextension and lateral motion, indicating ligamentous weakness.

There was undue mobility at the ankle joint, the lack of muscular resistance being striking. The same laxity was noted at the knee joint, with increased lateral mobility and a little rotary movement.

Fig. 3 (case 1).—Roentgenogram of the spinal column of patient, showing highly marked osteoporosis of all the vertebrae.

On palpation the muscles were distinctly hypotonic.

Neurologic Examination (Dr. L. Loeser).—The minimum response of the right facial nerve to the galvanic current was anodal closure contraction with 4 milliamperes and cathodal closure contraction with 5 milliamperes. The minimum response of the right ulnar nerve was anodal closure contraction with 3 milliamperes and cathodal closure contraction with 4 milliamperes. The minimum response of the right deep peroneal nerve (flexion of the foot) was anodal closure

contraction and cathodal closure contraction with 6 milliamperes (with much more forcible and greater contraction of the muscles of the foot on cathodal closure contraction than on anodal closure contraction, showing qualitatively the absence of degeneration of the nerve). The reactions indicated diminished muscular contractility due not to nerve degeneration but to muscle degeneration.

Roentgenographic Findings (Dr. J. Furst).—The sella turcica was comparatively small in the proportional transverse dimension (50 per cent undersized). The skull was of the ovoid type. The first anterior rib on both sides showed a moderate degree of porosity and a tendency to flaring. The extremities showed a marked degree of osteoporosis. This was especially observed at the cancellous ends of the long bones. The variation in structural detail at the lower end of the left femur was probably the end-result of a healed fracture at this point. A similar condition was observed at the intermediate third of the shaft of the right tibia.

The tibia and fibula of both sides showed a tendency to bowing.

The entire dorsal and lumbar parts of the spine showed a marked loss of calcium and poor definition of the medullary structure. In the lateral view the bodies of the vertebrae were concave. The fourth lumbar vertebra was almost in collapse. The dorsal part of the spine showed a moderate posterior curvature.

Laboratory Findings.—The urine was clear and had a specific gravity of 1.006. A blood count showed the following values: 71 per cent hemoglobin; 4,630,000 erythrocytes, and 8,000 leukocytes, with 50 per cent polymorphonuclears, 36 per cent lymphocytes, 5 per cent endothelial leukocytes, 6 per cent eosinophils, and 3 per cent basophils. There were slight anisocytosis and very slight poikilocytosis. The Wassermann, Kline and Kahn tests of the blood gave negative results. The calcium content of the blood was 10.1 mg. per hundred cubic centimeters; the phosphorus content was 3.8 mg., and the phosphatase content, 3.9 units (1 unit = 1 mg. of phosphatase liberated in one hour).

Calcium Balance (Six Day Test): The patient was kept on a special diet consisting of 60 Gm. of proteins, 33.5 Gm. of fats and 250 Gm. of carbohydrates. The daily intake of calcium was 0.121 Gm. Three glasses of water and 1 Gm. of sodium chloride were allowed daily. Carmine was used in order to delimit the beginning and end of the test. The calcium content was determined by the Kramer-Tisdall method; the phosphorus content, by the Benedict method. The results were as follows:

	Calcium, Gm. per 100 Cc.	Phosphorus Pentoxide, Gm. per 100 Cc.	Chlorides, Gm. per 100 Cc.
Urine	0.0933	5.496	23.1410
Feces	0.4980	0.251	0.0518
Total	0.5913	5.747	23.1988
Food	0.9540	1.261	2.5740

CASE 2.—*History.*—D. O., a white boy aged 15 months, had broken bones frequently, without traumatism. He was born spontaneously at home. He cried almost continuously as though in pain. His mother noticed that his legs were swollen. A physician informed her that the baby's legs and hips were fractured. The patient was kept in the hospital for six weeks, after which time the fractures were healed. Three or four months later a slight trauma produced a fracture of the left leg. In six weeks this leg healed. One or two months later, while the baby was being dressed, the left leg snapped again. This fracture healed well. Ten weeks before the patient was brought for examination, the mother, after carrying him a long distance, placed him gently on a counter, and the right leg broke. Three days later the patient suffered another fracture of the leg.

His appetite was poor, and there had been no regular gain in weight. There were two other children, both of whom were well and without any indication of a similar condition.

Physical Examination.—There was a definite deformity of the head, as though in passage through the birth canal there had been pressure in the frontal and occipital regions. There were dilated venules on the prominent forehead. The anterior fontanel admitted two fingers and was not bulging. The posterior fontanel was closed. There was loss of some hair in the occipital region. The head was enlarged, having an occipitofrontal circumference of 18 inches (45.7 cm.).

Eyes: The scleras showed definite but slight blue discoloration of equal hue. The lower lids were wrinkled.

Fig. 4 (case 2).—Clinical appearance of patient.

Mouth: The patient had a high-arched palate. Six upper and six lower teeth were present (delayed dentition). They were poorly developed and appeared decalcified.

Extremities: The right leg was shorter than the left. The distance from the anterior-superior spine to the internal malleolus measured 10½ inches (26.6 cm.) on the right side and 10 inches (25.4 cm.) on the left. The midsection of the right thigh measured 7 inches (17.8 cm.), and that of the left thigh, 8 inches (20.3 cm.). The left thigh showed anterior bowing and thickening.

Roentgenographic Findings (Dr. J. Furst).—Examination of the skull showed it to be of the hydrocephalic type and somewhat ovoid. The greatest increase in the skull's diameter was in the vertical oblique direction.

The upper extremities showed a moderate degree of absorption of lime salts, which had resulted in accentuation of the medulla and sharpening of the cortices, which probably contained increased amounts of lime salts.

The lower extremities showed a similar general appearance of the bones. There was evidence of old fractures of both femurs with a slight increase in their external bowing. More recent fractures were observed in the shaft of the tibia.

These findings suggested a systemic disturbance—osteoporosis imperfecta.

Fig. 5 (case 2).—Roentgenogram of the lower extremities of patient. In addition to generalized osteoporosis, evidence of eight new and old fractures is visible.

Laboratory Findings.—The urine was cloudy, with an acid reaction; the specific gravity was 1.024. The Wassermann, Kline and Kahn tests of the blood gave negative results. The calcium content of the blood was 11 mg. per hundred cubic centimeters; the phosphorus content was 3.4 mg. Two weeks later a second examination showed the calcium content to be 11.1 mg. and the phosphorus

content 4.7 mg. After another interval of two weeks the phosphorus content was 3.5 mg., and the phosphatase content, 4.9 units.

The calcium balance was estimated by the same methods as in case 1. The results were as follows:

	Calcium, Gm. per 100 Cc.	Phosphorus Pentoxide, Gm. per 100 Cc.	Chlorides, Gm. per 100 Cc.
Test of First Three Days			
Urine	0.011	0.408	0.5590
Feces	0.211	0.049	0.0743
Total	0.222	0.457	0.6333
Food	0.295	0.233	0.0618
Test of Second Three Days			
Urine	0.025	1.249	1.537
Feces	0.617	0.193	0.289
Total	0.642	1.442	1.826
Food	1.749	0.550	1.734

DIFFERENTIAL FACTORS

Our metabolic study strongly confirms the decided differences in the pathologic osseous conditions existing in hyperparathyroidism and those associated with blue scleras. Hyperactivity of the parathyroid glands results in a negative calcium and a negative phosphorus balance, with increased activity of phosphatase. This is in contradistinction to the findings in osteogenesis imperfecta, in which there is no hypercalcemia or hypophosphatemia and no increased excretion of calcium and phosphorus through the urine.

A careful perusal of the literature shows, furthermore, that in the cases of osteogenesis imperfecta and associated blue scleras in which an endocrine disturbance was assumed this assumption was based only on vague clinical symptoms, without consideration of the extremely important metabolic data and accurate investigation of the pathologic changes in the bones. Accordingly, various endocrine glands were suspected of being the causative factor. Kaznelson held the parathyroids responsible; Lesné, Hutinel and Dreyfus-Sée, and Sverdlov,[41] the thyroid, and Gutzeit, the hypophysis. Frocher mentioned changes in the anterior lobe of the pituitary as a direct cause and Schwarz, changes in the thymus, while Hein described an enlarged thymus. Casanovas pointed to hypofunction of the parathyroid glands or the thyroid, and Viallefont [42] and Terrien, to the parathyroid glands. On the other hand, Desoff expressed the opinion that the rather well established presence of hyperparathyroidism must be the source of the syndrome. These rather speculative assertions are, to say the least, not helpful in clarifying the relationship of the two conditions and are even misleading not only in the classification of the anomaly but in regard to therapeutic measures for, and operative intervention on, the suspected glands as well.

41. Sverdlov, G. O.: Zentralbl. f. d. ges. Ophth. **28**:75, 1933.
42. Viallefont, H.: Ann. d'ocul. **170**:289, 1933.

In considering both the similarities and the differences in the two conditions it should be borne in mind that osteogenesis imperfecta associated with blue scleras is characterized by dominant hereditary transmission accompanied by deafness. Two of the symptoms, the blue scleras and the fragile bones, are present at birth. Osteitis fibrosa cystica is likewise a generalized disease of the entire skeleton, leading to numerous spontaneous fractures, but appears at a much later stage of life. Hunter and Turnbull considered the usual age for its appearance to be from 30 to 35 years. Gutman, Swenson and Parsons,[43] in analyzing one hundred and fifteen cases of hyperparathyroidism according to the patients' ages at the time of operation or death, found no patients in the first decade of life, seven in the second, seventeen in the third, nineteen in the fourth, twenty-eight in the fifth, twenty-three in the sixth and five in the seventh. Age is therefore an important clinical factor in the evaluation of the causes of spontaneous fractures. In osteogenesis imperfecta the fractures are intra-uterine or acquired in early life, while in osteitis fibrosa cystica they occur at a later age, a period when in the former condition no further pathologic changes take place.

In the course of osteogenesis imperfecta hereditary deafness is likely to develop after the age of 20, being caused, according to the roentgenographic findings of Stenvers,[44] by structural changes in the petrous bone and by calcification of the labyrinth. The pathologic osseous changes in osteogenesis imperfecta were outlined by Albright, Aub and Bauer [15a] as a depression in bone formation combined with normal absorption, with no increase in osteoclasts or in fibrosis. It is well to point out again that this disease is due to congenital faulty differentiation or malformation of the mesenchyma, owing to which the osteoblasts either are lacking or are unable to perform their allotted physiologic function, the end-result being the formation of fibrous bone tissue instead of further development into shell bone. There is no active mobilization of the calcium supply in the body, and consequently there is no negative calcium or phosphorus balance and no increase in phosphatase activity. The spontaneous multiple fractures and the prompt healing without considerable callus formation manifest themselves in both conditions but are based on entirely different pathologic principles. In contrast to the observations in cases of osteogenesis imperfecta, in those of osteitis fibrosa cystica there are typical laboratory findings showing the active mobilization of calcium and phosphorus. This results in the unique combination of a negative balance of these compounds

43. Gutman, A. B.; Swenson, P. C., and Parsons, W. B.: The Differential Diagnosis of Hyperparathyroidism, J. A. M. A. **103**:87 (July 14) 1934.

44. Stenvers, H. W.: Arch. f. Ophth. **95**:94, 1918.

with an increased blood phosphatase content, changes which are caused by overproduction of the active principle of the parathyroid glands. The metabolic alterations are usually marked and show a dramatic change following removal of the tumor almost invariably found in hyperparathyroidism, with associated elimination of the excessive stimulus of osteoclastic resorption.

The underlying cause of the excessive excretion of calcium and phosphorus is generally adenoma of the parathyroid glands (the adenoma being benign only in relation to its morphology). Some authors, like Gold, prefer to follow the classification of struma instead of tumors and use the term struma parathyroidea diffusa or nodosa parenchymatosa.

The adenomas are of minute size and show increased vascularization and decrease in, or absence of, the fat cells. The principal cells are mostly considered as functionally essential to the secretion of the gland, but opinions differ widely as to which types of the chief cells are the important ones. Petersen and Schall considered the less chromatic types as important; Getzova and Hartwich, the darker red ones. On the other hand, Koenigstein and Forsyth regarded the oxyphil cells as the active and colloid-producing types. The number of cells seems to increase with the age of the patient, a factor probably associated with the amount of colloid and fat deposited. Some observers even entertained the idea that the principal and oxyphil cells are possibly only different morphologic modifications representing various stages of the physiologic function. In their series of instances in which a single adenoma occurred Albright, Bloomberg, Castelman and Churchill [15c] noted that the tumor consisted of slightly enlarged hyperchromatic chief cells without mitosis, the cells being arranged in columnar or cylindric formation. In addition to these, cells with water-clear cytoplasm (Getzova) and oxyphil cells were present, the former rarely comprising the major part of the specimen.

The single adenoma or, possibly, multiple adenomas of the gland are doubtlessly responsible for the pathologic picture of parathyroid hyperactivity. Rarely, however, does one encounter cases with the classic metabolic findings of hyperparathyroidism in which, instead of adenomas, hyperplasia of all the parathyroid tissues is common. When hyperplasia is present, according to Albright and his co-workers, the water-clear cells predominate; they are of a much larger size than those encountered in the single tumors. The hyperplasia of the thyroid glands, in conjunction with the metabolic picture of hyperparathyroidism, is considered as forming an important entity and suggests the pituitary gland as the source of stimulation. This pathologic picture must be borne in mind in order to differentiate this form of hyperparathyroidism from the "hypertrophy of Erdheim" of the parathyroid glands in condi-

tions such as osteomalacia and rickets. In the latter, hyperplasia represents an inadequate attempt at compensation, the alteration being the result and not the cause of the disease. Primarily, hyperfunction and dysfunction are established in the sense of regulatory function, which induces hyperplasia of a correlative regulatory character and by overtaxation causes pathogenic action on the skeleton.

PRESENCE OF BLUE SCLERAS IN HYPERPARATHYROIDISM

A study of the literature of known and proved cases of hyperparathyroidism (associated with adenomas and hyperplasias) yields little about blue scleras. In more than one hundred cases we could find only four instances in which blue scleras were noticed in connection with distinct parathyroid hyperactivity. These cases are briefly summarized.

Wichmann [45] reported a typical case of a woman 45 years of age on whom an exact determination of the calcium metabolism was made. The scleras were described as mildly bluish. In the first of two cases of parathyroid tumor cited by Hanke,[46] a woman aged 33 and her mother both showed blue scleras. The subjective complaints began at the age of 18, and she had her first spontaneous fracture at the age of 27. The patient exhibited typical hyperparathyroidism; operation proved the presence of adenomas. Similarly, Cohen and Kelly [47] reported the case of a woman 48 years of age who showed swelling of the jaw at the age of 39, associated with blue scleras. She presented the clinical picture of hyperparathyroidism—a negative calcium balance and a tumor of the right inferior parathyroid gland. Keynes and Taylor [48] noticed brilliantly blue scleras in a man who suffered from osteoclastoma of the right maxilla, muscular atony, spontaneous fracture of the right humerus and generalized typical pathologic osseous changes caused by a large tumor of the left parathyroid gland. In all these four cases the presence of blue scleras was observed. In Hanke's case even hereditary transmission was mentioned.

It would be tedious to enumerate all the classic symptoms of parathyroid hyperactivity in these four cases, as they were numerous. Suffice it to say that blue scleras were present in intimate association with typical parathyroid disorder. We must emphasize, however, that the clinical picture, the occurrence of fractures at a rather later period of life, is more characteristic of parathyroid disorder than of the symptom complex of osteogenesis imperfecta. These instances stand in marked contradistinction to the occurrence of blue scleras with osteoporosis and

45. Wichmann, F. W.: Deutsche Ztschr. f. Chir. **235**:619, 1932.
46. Hanke, H.: Arch. f. klin. Chir. **172**:366, 1932.
47. Cohen, H., and Kelly, R. E.: Brit. J. Surg. **20**:472, 1933.
48. Keynes, G., and Taylor, H.: Brit. J. Surg. **21**:20, 1933.

deafness but with absence of parathyroid involvement. Our two cases herein reported, in which the calcium and phosphorus balances were carefully determined, illustrate the fact clearly.

On the basis of the present-day knowledge, an explanation of the simultaneous presence of hyperparathyroidism and blue scleras in the four reported cases is still lacking. There is the possibility of the coexistence of two different pathologic entities, a congenital syndrome of blue scleras and superimposed parathyroid tumor formation, causing the typical negative calcium and negative phosphorus balance and the pathologic osseous condition, which are never encountered together in association with the congenital form of blue scleras alone.

As an item of interest mention may be made of Albers-Schönberg's disease, or osteopetrosis, which roentgenographically and pathologically may be regarded as diametrically different from the osteoporosis associated with blue scleras. In cases of marble bones there are a striking familial tendency and, occasionally, a hereditary influence. McCune and Bradley [49] thought that osteopetrosis is a true developmental disease traceable to faulty differentiation of the primitive common forerunner of osteogenic and hematogenic tissue, the ultimate cause being inherent in the germ plasm. There is no evidence at present to suggest any endocrine disturbance. The inconstant changes in the calcium and the phosphorus metabolism that occur occasionally in cases of osteogenesis imperfecta and osteopetrosis are probably only secondary disturbances due to the primary disorder of the bone formation in this exceedingly rare anomaly.

CONCLUSIONS

In the usual cases of blue scleras associated with spontaneous fractures and hereditary deafness there are not sufficient variations in the values for calcium, phosphorus and phosphatase to warrant a supposition of involved endocrine disturbance.

In osteogenesis imperfecta there is evidence of decided hereditary transmission, in contrast to hyperparathyroidism, which shows no such influence.

Osteitis fibrosa cystica is based on hyperactivity of the parathyroid glands, the usual cause being tumor formation within the glands, resulting in mobilization of the supply of calcium. The spontaneous fractures occur in adult life and are accompanied by a negative calcium balance, cyst formation and giant cell tumors of the bones.

The small minority of cases in which blue scleras are associated with proved parathyroid disorder are rare exceptions, the pathologic

49. McCune, D. J., and Bradley, C.: Osteopetrosis (Marble Bones) in an Infant; Review of the Literature and Report of a Case, Am. J. Dis. Child. **48**:949 (Nov.) 1934.

features not constituting the usual picture. The most plausible explanation lies in the possibility that the condition of hyperparathyroidism has been superimposed on a preexistent congenital anomaly.

Similarly, spontaneous fractures are clinical features of the other form of generalized osseous disease, osteogenesis imperfecta, associated with blue scleras and deafness. They occur in utero or in infancy or childhood, in contradistinction to the adult age incidence of hyperparathyroidism. The roentgenographic picture of the osseous condition is that of osteoporosis. There are absence of a negative calcium balance and lack of increased phosphatase activity in osteogenesis imperfecta, the latter being a distinguishing feature of the parathyroid syndrome. Furthermore, blue scleras are characterized by dominant hereditary transmission and are due to a congenital faulty differentiation or malformation of the mesenchyma.

PARTIAL RUPTURE OF THE LAMINA CRIBROSA FROM CONTUSION OF THE EYEBALL

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Injury of the optic nerve in the scleral canal is rare and usually results from a penetrating wound of the eye (Parsons [1]). Clinical cases of indirect rupture of the lamina cribrosa have been recorded by Lang,[2] Cramer [3] and Gonin.[4] As the eyeball was not removed in these cases, microscopic descriptions are lacking. Wagenmann [5] did not mention indirect rupture of the lamina cribrosa in the *Handbuch der gesamten Augenheilkunde* (von Graefe and Saemisch), nor did von Szily [6] in his atlas of war injuries. Rönne [7] considered that "evulsion of the optic nerve" had been described from twenty to twenty-five times but that it must in all probability be much more common. Only five of the instances which he collected from the literature had been caused by indirect injury. Lister,[8] discussing concussion changes met with in military practice, suggested the name "expulsion of the optic nerve" for the injury which is caused by contusion of the ocular globe.

A report of the microscopic observations in two cases follows. In both instances the eyeball was removed because of painful traumatic glaucoma some time after the injury, a severe contusion of the eyeball. In neither case could the condition of the optic nerve and lamina cribrosa be examined clinically because of hemorrhage into the vitreous.

From the Department of Ophthalmology of the University of Amsterdam (Prof. W. P. C. Zeeman, M.D.).

1. Parsons, J. H.: The Pathology of the Eye, London, Hodder & Stoughton, 1908.

2. Lang, W.: Rupture of the Lamina Cribrosa and Optic Nerve-Fibers at the Papilla, Tr. Ophth. Soc. U. Kingdom **21**:98, 1901.

3. Cramer, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 4.

4. Gonin, J.: Ruptures partielles de la papille optique, Ann. d'ocul. **147**: 16, 1912.

5. Wagenmann, A.: Die Verletzungen der Auges, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 3, Leipzig, Wilhelm Engelmann, 1915.

6. von Szily, A.: Atlas der Kreigsaugenheilkunde, ed. 3, Stuttgart, Ferdinand Enke, 1918.

7. Rönne, H., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5.

8. Lister, W.: Some Concussion Changes Met with in Military Practice, Brit. J. Ophth. **8**:305, 1924.

In both cases the microscope showed also an atypical, incomplete rupture of the sclera. This type of rupture is new to the literature and will be described in a separate article.[9] The serious lesions in the retina, choroid, ciliary vessels and nerves will be described in an article on traumatic glaucoma.

REPORT OF MICROSCOPIC OBSERVATIONS IN TWO CASES

CASE 1 (fig. 1).—The optic nerve exhibited a very deep funnel-shaped excavation which ran through the lamina cribrosa to the exterior of the eyeball. As the nerve had been cut rather short in the operation for removal of the eyeball, it was impossible to determine how far the excavation had continued into the nerve.

Fig. 1 (case 1).—Rupture of the lamina cribrosa and optic nerve.

The lamina cribrosa had been bent backward and at its center exhibited a large defect through which the optic cup passed outward. This cup differed from a glaucomatous cup by its unusual shape and by the direction of the anterior fibers of the lamina cribrosa. Moreover, glia and nerve tissue were seen anterior to the lamina. The posterior fibers of the lamina had been severed in the operation. The anterior fibers, however, were clearly seen in the microscopic sections and appeared to have been straightened in a posterior direction. Anterior to them the columns of optic nerve fibers gave the impression of having been displaced backward.

CASE 2 (fig. 2).—The optic nerve exhibited a markedly glaucomatous cup and a few caverns. It was also partly atrophic, as was demonstrated by staining the myelin sheaths.

9. Tillema, A.: Atypical, Indirect, Incomplete Rupture of the Sclera, Brit. J. Ophth. **20**:193, 1936.

Lateral to the central vein an irregularly sausage-shaped cavity was seen which opened anteriorly into the vitreous body and posteriorly into the central vein. It was not lined with endothelium but was filled with a loose tissue derived probably from the vitreous and containing a fair amount of connective tissue. Cells with a large oval pale nucleus and cells with a rather dark oval nucleus (probably fibroblasts) were scattered irregularly throughout this tissue. In a few places young vessels were entering. Some of the cells contained fine pigment granules. This mass of tissue projected posteriorly into the lumen of the central vein.

Medial to this cavity the central vein was found, and medial to the vein, the artery. The latter showed an uninterrupted course from the optic nerve into the retina. The vein, however, was interrupted. Coming from the retina and turning posteriorly along the floor of the glaucomatous optic cup it was still filled with red blood corpuscles. Close to the cavity it made a sharp turn in a posterior

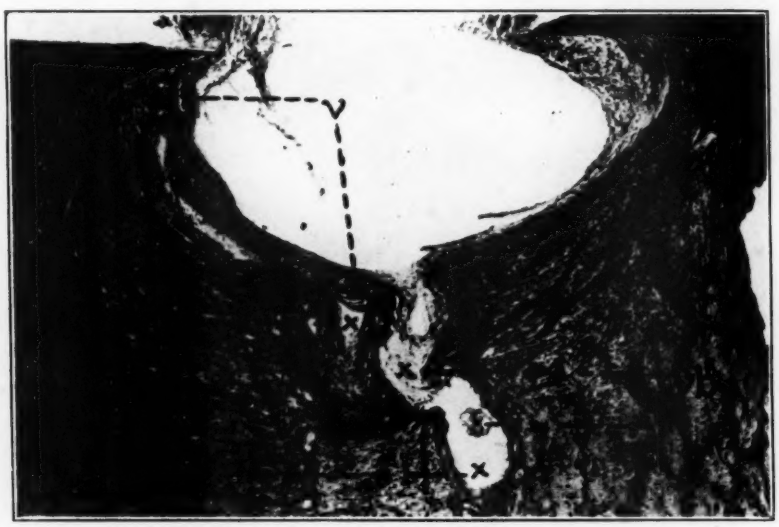

Fig. 2 (case 2).—Rupture of the lamina cribrosa and central vein: *a*, central artery; *ct*, young connective tissue; *v*, central vein; *vb*, vitreous body.

direction and ended as a delicate tube of endothelial cells on the medial wall of the cavity. Farther back behind the lamina cribrosa one picked it up again, containing only a few white blood cells. Much farther back it received many small tributaries and was full once more. It will be noted from this description that the central vein and the cavity appeared side by side anteriorly but that they continued as one structure posteriorly.

In the region of the lamina cribrosa the vein, artery and cavity were surrounded by a mass of fairly dense irregular connective tissue. This tissue was very cellular and contained blood capillaries. Like the tissue filling the cavity, it contained a few pigment granules here and there. From the lamina cribrosa it continued backward, surrounding the central vein and containing a large number of lymphocytes. A little farther back it faded out.

Inasmuch as no junction of veins was found in the optic nerve, the superior and inferior central veins must have joined in the retina.

Continuation of the optic cup backward is found both as a congenital anomaly (Hagedoorn [10]) and is a feature of glaucoma (Elschnig [11]). In both instances the condition vaguely resembles the prolapse of the vitreous described in a foregoing paragraph. The rupture of the central vein, however, and the presence of a mass of young connective tissue containing pigment granules leave no doubt as to the traumatic origin of the condition that has been described.

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10. Hagedoorn, A.: Ein anatomischer Beitrag zur Frage der umschriebenen Grubenbildung am Sehnerveneintritt, Arch. f. Augenh. **99**:387, 1928.

11. Elschnig, A.: Augenärztliche Unterrichtstafeln, Breslau, J. V. Kern, 1900; in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11.

HUMAN CYCLOPIA

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The ophthalmologic clinic of the Northwestern University has received from the obstetric department of the Michael Reese Hospital, through the kindness of Dr. Harry Gradle, the head of a cyclopic fetus, concerning which, because of its rarity, I wish to make a report. To show the variation in degree of singleness in these cases, descriptions of two other cyclops from the Anatomic Museum of the Northwestern University Medical School have been included. The first of these (fig. 1) had a single median orbit, four lids forming a single diamond-shaped palpebral fissure in which were seen two bulbs of about normal size for its age, each with its cornea 5 mm. in diameter. The two bulbs fused posteriorly in the midline. Above the orbit was a proboscis 1 by 1.5 cm. No other deformity of the head was apparent. The second cyclops (fig. 2) had a single median orbit similar to the first, containing a single eye slightly larger than normal, with a single horizontally oval cornea measuring 4 by 7 mm. Above this was a proboscis 1 cm. in diameter and 2 cm. long. The third specimen (fig. 3), which is the subject of this report, had a deep horizontal furrow below the proboscis, and deep in the orbit was a microphthalmic eye which was not visible from the outside. (The lower part of the head in figure 3 had been cut off and a cut made through the skin above the proboscis.)

REPORT OF A CASE

The history in the third case was as follows:

Mrs. W., the mother, had one normal child, 5 years of age. During the third, fourth and fifth months of the present pregnancy there had been considerable nausea and vomiting, slight edema and occasional dysuria. The urine showed a trace of albumin, which persisted in spite of a diet poor in salt. The blood chemistry was normal. Physical examination showed a blood pressure of 168 systolic and 96 diastolic and a moderate enlargement of the thyroid gland. At nine months, on Oct. 3, 1930, a fetus weighing 1,440 Gm. was born. It had a single orbit with a proboscis above; the left hand had six fingers, and the genitalia were incompletely developed male organs. In 1934 Mrs. W. delivered a normal healthy child after a normal pregnancy. There have been no other pregnancies.

The head of this fetus was preserved in a solution of formaldehyde and was received at the laboratory with the lower part removed. It measured 9 cm. anteroposteriorly and 7 cm. bitemporally. Across the face was a horizontal furrow

From the Department of Ophthalmology, the Northwestern University Medical School.

which showed histologically normal lid structures. Some tissue, later found to be conjunctival tissue, protruded from the center of the furrow. Above this was the proboscis. No other gross deformities were seen. The head was decalcified, embedded in pyroxylin and sectioned from above downward, and every tenth section through the region of the eye was stained with hematoxylin and eosin. The orbit

Fig. 1.—A cyclops from the Anatomic Museum of the Northwestern University Medical School.

Fig. 2.—A cyclops from the Anatomic Museum of the Northwestern University Medical School.

was 25 mm. wide and was filled with fatty areolar tissue in which were two sets of muscles; the small eye was placed superiorly and posteriorly in the orbit. Anteriorly there were a narrow conjunctival sac and lids, and in the center above the lids was the proboscis.

The eye itself consisted of two anterior segments which fused into a single posterior segment from which a single optic nerve ran posteriorly and superiorly into the skull through a bony canal 7 mm. wide. The two anterior segments were unequal in size, the right being 8 mm. in width at its greatest single width and the left 5 mm. in width. The single posterior part was 4 mm. wide. The tunica fibrosa (fig. 4, *1*) was a dense fibrous tissue very similar to normal sclera. No differentiation of a cornea could be made out anteriorly, except that the conjunctival sac came in contact with the right half in one place (fig. 4, *2*). This layer formed the two anterior segments and fused medially into a single wall, which posteriorly became a thicker mass and extended into each cavity to form a stalk around which the detached retina (fig. 4, *5*) and the lens were pressed, leaving little vitreous (fig. 4, *10*) but a large cavity anteriorly in the right and a lesser one in the left. In this thicker mass extending into the eye was a sharply

Fig. 3.—The cyclops reported on here.

outlined oval plate of cartilage (fig. 4, *4*). In the right cavity the cartilage was 2.5 mm. long, and in the left, about 1.5 mm. The right side contained some vascular granulation tissue (fig. 4, *6*) just posterior to the lens. In this area was deposited some calcium. The mass in the right side was adherent to the choroid anteriorly and to the retina on one side (fig. 4, *7*). The choroid was a thin layer and contained little blood, only a few medium-sized vessels being visible. Chromatophores were present. Anteriorly, it became difficult to distinguish this from the tunica fibrosa; in one place a thickening was seen which was probably ciliary body and a small bit of iris stroma (fig. 4, *8*). A normal layer of pigment (fig. 4, *9*) lay in contact with the fibrous layer lining the whole cavity. Where the iris tissue was seen in the right side, a small projection extended posteriorly into the cavity, and from this two fine blood vessels (fig. 4, *11*) extended posteriorly to the lens.

Fig. 4.—Photomicrograph of the eye: *1*, the sclera; *2*, the cornea; *3*, the lens; *4*, cartilage; *5*, the retina; *6*, granulation tissue; *7*, the choroid; *8*, the iris; *9*, pigment; *10*, the vitreous; *11*, blood vessels; *12*, the conjunctival sac, and *13*, the lid.

Fig. 5.—Photomicrograph showing the single optic nerve.

The lens (fig. 4, *3*) on each side was in contact with the connective tissue mass posterior to it. The right lens was 3 mm. and the left lens 1 mm. in diameter. The capsule and the epithelium of the anterior surface were normal. The substance of the left lens was homogeneous; that of the right was split up irregularly. Anterior to the right lens was a cavity filled with light pink homogeneous fluid. This cavity comprised half of the interior of the bulb. Anterior to the left lens was a smaller cavity. Posterior to the right lens was folded retina (fig. 4, *5*) on one side of the lens, and on the other side retina that was smooth (though away from the wall), probably separated during fixation. Within the latter a small amount of vitreous (fig. 4, *10*) was present. In the folded part were distinct rosettes. The retina showed two cells layers and in places a ganglion cell layer. The external limiting membrane was present, but there were no rods or cones. In the left half of the eye the retina was not as much folded, but lay more smoothly and more nearly in contact with the walls of the cavity. In several places in the granulation tissue back of the lens and just outside of the sclera were occasional groups of polymorphonuclear cells.

Farther posterior and superior there was a fusion of the two cavities, so that only a single cavity lined with retina was present, and from this the single optic nerve ran posteriorly (fig. 5). This was stained with the Davenport stain for nerve fibers and showed normal young nerve fibers in the optic nerve. No sign of a chiasm was present. The nerve was followed 2 cm. behind the eye but could not be found farther back.

COMMENT

Cases of cyclopia have been known since very early, as indicated by the story of the cyclops in Ulysses. In a textbook on ophthalmology published in 1830, Rosas [1] said: "There are many reports of cyclopic eyes." Twenty-three cases of cyclopia in man were found reported in the literature available. The condition has been reported in horses, birds, rats and fish and is especially frequent in pigs. Although there is great variation in the degree of deformity, the usual picture is partial or complete fusion of the two eyes in the midline with a proboscis above instead of a nose and defects in the forebrain, such as absence of the first cranial nerve and sometimes of the second. Other defects may be present, such as polydactylism, anencephaly and astomatism.

About 1910 there was considerable interest in this condition, especially in the theories as to its cause. These were well presented by Hill.[2] About the same time Stockard [3] reported his experiments on salamander eggs, in which normal eggs produced 50 per cent cyclopic embryos when the magnesium chloride content of the water in which

1. Rosas, cited by Nieden, A.: Ueber Anophthalmia cyclopica, Arch. f. Augenh. **22**:61, 1890-1891.

2. Hill, E.: Cyclopia, Its Bearing upon Certain Problems of Teratogenesis and of Normal Embryology, Arch. Ophth. **49**:597, 1920.

3. Stockard, C. R.: The Artificial Production of One-Eyed Monsters and Other Defects, Which Occur in Nature, by the Use of Chemicals, Anat. Rec. **3**:167, 1909.

they were developing was increased. Stockard expressed the belief that the magnesium had an inhibitory or anesthetic effect on the out-pushing and separation of the optic vesicles. He stated that the eye primordium is in the midventral line of the brain and that early arrest of development causes this to remain median or divide in part to form the hourglass-shaped eye. Wolff [4] has produced cyclopia in chicks by irradiation at an early stage.

An interesting point is the frequent finding of a single posterior part and a double anterior segment of the eye. In twelve of sixteen cases in which the interior of the eye was described, there were two lenses but a single optic nerve. It seems probable that the single optic vesicle stimulated the surface ectoderm on each side of the head, causing the formation of two lenses which conditioned the formation of the other structures of the anterior segment about the lenses, making an apparent two eyes from the initial single vesicle. This would fit in well with the theory that the eyes come from a single median anlage which separates normally into two, but that in cyclopic eyes this separation fails to take place, rather than with the theory that there are originally two separate anlagen which in cyclopic monsters become fused. This has been a point of dispute.

Another interesting finding is the rosettes of the retina. This was mentioned by Hill [2] in his case report, but he found by staining with a special stain for neuroglia that there was only a superficial resemblance. Rosettes are due to excessive growth of the primitive outer neuroblastic layer which furnishes nuclei of cones and rods and becomes the outer nuclear layer. Goldstein and Wexler [5] found such rosettes in embryos in cases in which roentgen irradiation had been used to terminate pregnancy during the second month.

The finding of cartilage in the eye in cyclopean eyes has been mentioned in the literature. Certain reptiles have plates of cartilage in the eye. The significance of this in the case reported here is not clear. The cartilage may be the beginning of the bone formation that occurs in shrunken or degenerated eyes. The granulation tissue and the few polymorphonuclears indicate that the early injury which this embryo sustained was probably an infection rather than a chemical or physical injury.

122 South Michigan Avenue.

4. Wolff, E.: Recherches expérimentales sur la cyclopie, Arch. d'anat. d'histol. et d'embryol. **18**:145, 1934.

5. Goldstein, I., and Wexler, D.: Rosette Formation in Eyes of Irradiated Human Embryos, Tr. Am. Acad. Ophth. **35**:140, 1930.

MELANOCARCINOMA OF THE CONJUNCTIVA

REPORT OF A CASE

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Epithelioma of the conjunctiva may vary in malignancy, but in about 80 per cent of the cases it tends to recur, even when superficial. If the diagnosis is made early, it is proper to remove the tumor; but if the eyeball itself is involved, complete enucleation of the globe, together with exenteration of the remainder of the orbital contents, is indicated. Some form of radiation therapy should be employed after following surgical intervention.

Malignant growths in the bulbar conjunctiva are often the result of extension from a primary focus in the conjunctiva of the lid. This primary focus may be a nevus, which is a congenital growth usually considered benign. It is a smooth, flat tumor highly pigmented with melanin. The color may vary from light to dark red-brown. These nevi of the conjunctiva have much the same structure histologically as that of the ordinary pigmented nevi found on the skin elsewhere on the body. Nests of pigment-bearing cells lie in a network of connective tissue, with conjunctival epithelium penetrating into the structure. Several theories as to the origin of these nevus cells have been advanced. We may mention here: (1) the theory that they derive from the endothelial cells of the lymph vessels, as was advocated by Recklinghausen; (2) the view that they arise from the surface epithelium, as was set forth by Unna,[1] and (3) Ribbert's contention that they come from the chromatophores, which are situated in the delicate connective tissue of the conjunctiva. Bloch,[2] in a brilliant piece of chemical research, developed the so-called dopa reaction, wherein he showed that the epidermal cells alone produce melanin in normal tissues. D'Agata[3] later showed that the only cases in which connective tissue or endothelial cells could be shown to be melanin-producing by the dopa reaction were those of tumor. It is felt by most workers today that pigmented nevus cells arise from the epithelium. Hence pigmented tumors that are

1. Unna, P. G.: Berl. klin. Wchnschr. **30**:14, 1893.
2. Bloch, B.: Ztschr. f. phys. Chem. **98**:226, 1916-1917.
3. D'Agata, G.: Tumori **9**:121, 1922.

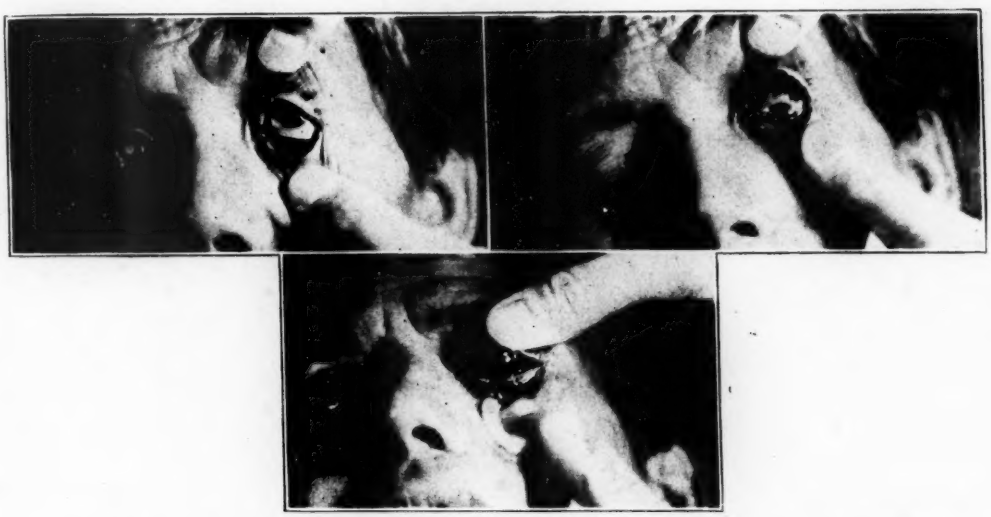

Fig. 1.—Photograph of the patient. *A* shows invasion of conjunctiva of the left lower lid by melanocarcinoma; *B*, involvement of the bulbar conjunctiva, and *C*, involvement of the conjunctiva, with the left upper lid everted.

Fig. 2.—Microscopic section of the melanocarcinoma; low power magnification.

derived from pigmented nevi would more properly be called melanocarcinomas rather than sarcomas.

Melanocarcinoma shows no marked tendency to penetrate the eyeball in its early stages. Later it may grow over the cornea, thus impairing vision. The entire conjunctival sac may be invaded. Frequent, alarming hemorrhages often occur. Metastases sooner or later end the patient's life.

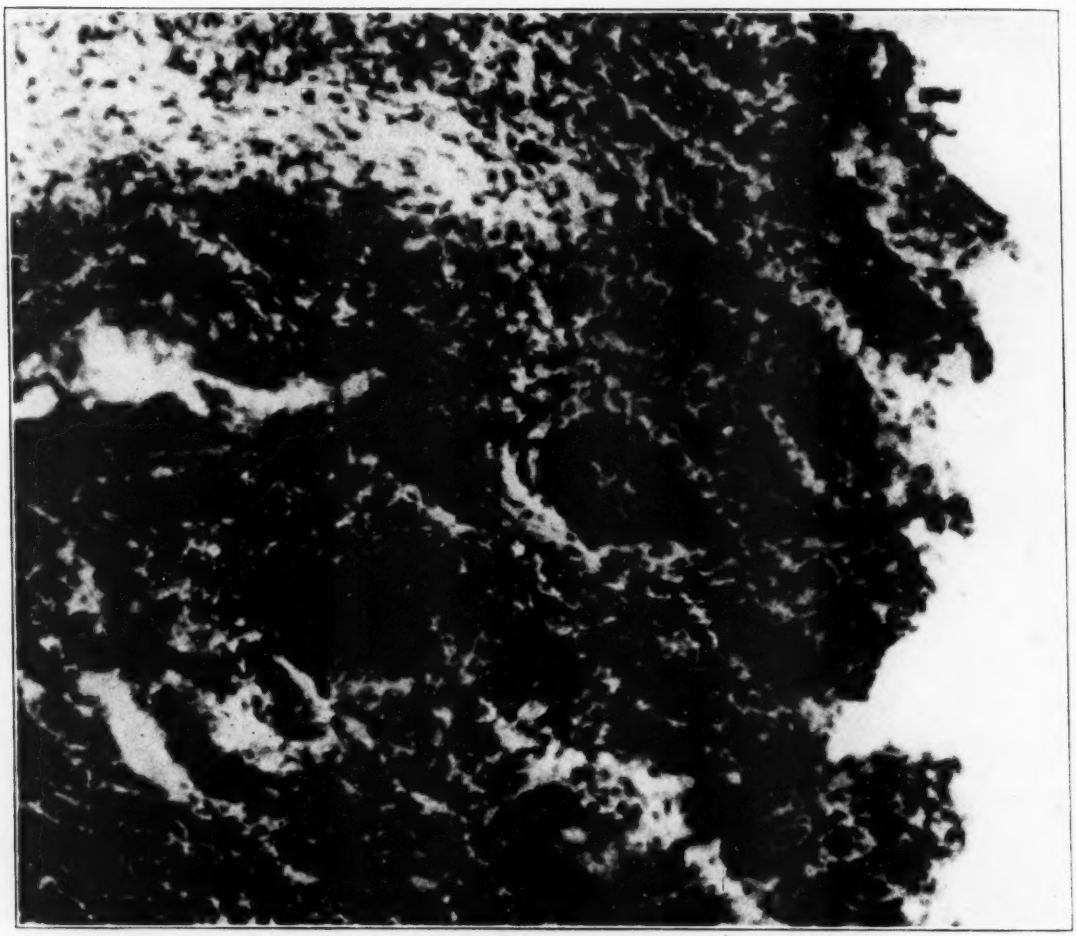

Fig. 3.—Microscopic section of the melanocarcinoma; high power magnification.

The diagnosis of melanocarcinoma is made from (*a*) the history, (*b*) the clinical examination, (*c*) biopsy of sections and of pathologic tissue and (*d*) the dopa reaction.

We herewith report a case of melanocarcinoma involving the greater portion of the palpebral conjunctiva of the left eye and extending to the bulbar conjunctiva. This malignant growth seemingly arose from

a pigmented nevus on the internal conjunctival surface of the upper lid of the involved eye.

REPORT OF CASE

I. E., a Mexican widow, aged 50, presented herself for consultation on Oct. 22, 1935. Her complaints were: (1) itching of the left eyeball and lids, (2) excessive watering of the left eye, (3) a slight bloody discharge from the left eye and (4) a small dark brown mass projecting from the under part of the left upper lid.

Six months previously the patient began to notice an increasing itching sensation of the left eye and lids. There was an unusually copious flow of tears. While facing a mirror one day she noticed a small dark brown spot on the left upper lid, behind the lashes. She turned the lid up slightly and noticed a dark substance covering a portion of the inner surface of this lid. Treatment consisting of frequent irrigations of the eye with water was instituted in the home. About one month later she first noticed that the tears were often slightly blood-tinged. As time passed she found that her pillows were blood-stained when she awoke in the morning.

Fig. 4.—Photograph of the patient taken on Jan. 21, 1936, showing definite progress of the tumor and enlargement of the anterior preauricular glands.

Examination on October 22 revealed a rather emaciated, apprehensive middle-aged woman. The right eye presented an old leukoma of the cornea that completely covered the pupil. Vision in this eye was but questionable perception of light. An internal squint was present. Examination of the left eye showed vision of 1.2. In this eye there were no squint, no abnormal position of the lids, no ulceration of the margins of the lids and no inflammation of the lacrimal apparatus. The cornea was clear. The palpebral conjunctiva of both lids exhibited several isolated patches of a dark brown, friable substance. Hemorrhage followed attempts to dislodge this material. One patch on the upper lid was the largest, being about 0.5 cm. in diameter. The temporal aspect of the bulbar conjunctiva exhibited, near the superior fornix, several round areas of the same dark brown substance, none of which was larger than 0.1 cm. in diameter. The intra-ocular tension of the eye was not increased. The pupil was regular and active to light and in accommodation. The media were clear. The fundus showed no pathologic changes.

A pathologist was called in conference. He examined a section of the suspected material and gave an opinion that the condition was melanocarcinoma. A roentgonologist was consulted. He felt that the condition was too far advanced to be helped by radiation therapy.

Mild astringent eye drops were prescribed, and the patient was requested to return at intervals.

On November 6 the patient was seen by us for the second time. The growth had progressed little. She was experiencing no pain. On November 15 she complained of intermittent sharp pains in the left eyeball, frequent severe frontal headaches, transient dimness of vision and a burning sensation in the left eyelids and the nose. At this time the growth under the left upper lid had begun to pile up in such fashion that the lid was being markedly displaced outward. The areas of the growth that were once discrete had become coalesced. Little of the palpebral conjunctiva remained uninvolved. The nasal half of the bulbar conjunctiva and the cornea were clear. The patient mentioned during this visit that she had had several mild hemorrhages from the left side of the nose. Examination of the nose revealed no gross signs of metastasis, however.

Since (*a*) this patient was totally blind in the right eye, (*b*) the disease process of the left eye was too far advanced for radiation therapy to be of help, (*c*) total exenteration of the left orbit was probably the only choice remaining and (*d*) there was a possibility that metastases had already progressed by extension down the left nasolacrimal duct, we were compelled to adopt a highly unsatisfactory program of treatment, that is, treatment solely palliative in intent.

SUMMARY

A case is reported of melanocarcinoma of the palpebral and bulbar conjunctiva with possible metastasis by extension down the nasolacrimal duct.

FILTRATION EXPERIMENTS WITH THE VIRUS OF INCLUSION BLENNORRHEA

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AND
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The evidence for the etiologic significance of certain morphologic elements (elementary and initial bodies) observed constantly in the cytoplasm of the epithelial cells of the conjunctiva in inclusion blennorrhea of the new-born has recently been strengthened by the experiments of Thygeson,[1] who reported his investigation in eleven cases of the disease. His statement that these inclusions are the causal agents of inclusion conjunctivitis is based on (1) their constant presence, (2) their absence in bacterial conjunctivitis, (3) the absence of pathogenic bacteria in inclusion blennorrhea, (4) the transmissibility of inclusion conjunctivitis and the presence of inclusions in the new host and (5) the production of the disease with bacteria-free suspensions of epithelial scrapings containing inclusions.

Two patients with inclusion blennorrhea were available to us for detailed study and inoculation of animals, and the positive results obtained seem to justify their being reported here. The graded collodion membranes [2] used in our experiments were prepared by the method described in detail by Bauer and Hughes.[3] The material for filtration was prepared as described by Thygeson, and the smears stained with Giemsa's solution were decolorized slightly, as he suggested, in order to facilitate the differentiation of the inclusion bodies from leukocytic granules. The filtrations were carried out under a 50 pound (22.7 Kg.) pressure (nitrogen) in the Bauer and Hughes filter chamber.[4] The suspensions were first freed from particles of tissue by centrifugation; hence, the clogging of the membrane was reduced to the minimum, and nearly the whole of the fluid was recovered in the filtrate. The protocols for the two experiments follow.

From the Departments of Research Bacteriology and Ophthalmology, the Northwestern University Medical School.

1. Thygeson, P.: Am. J. Ophth. **17**:1019, 1934
2. Elford, W. J.: J. Path. & Bact. **34**:505, 1931.
3. Bauer, J. H., and Hughes, T. P.: J. Gen. Physiol. **18**:143, 1934.
4. Dr. Bauer had one of these filter chambers made for us.

EXPERIMENTAL INVESTIGATION

EXPERIMENT 1.—In A. S. conjunctivitis developed thirteen days after birth. Examination of smears and scrapings failed to show gonococci or other bacteria but revealed many typical inclusions. Material for experimentation was taken thirty-one days after birth by gentle removal with a platinum spatula of epithelium from the conjunctiva of the upper and lower lids of both eyes. The material was suspended in 2 cc. of plain infusion broth; the shreds were broken by grinding in a mortar, and the tissue débris was removed by centrifugation for ten minutes at low speed. Six drops of the clear supernatant fluid was instilled into the right eye of baboon 1 (sphinx baboon, Papio sphinx) after preliminary scarification of the conjunctiva of the upper and lower lids. The remainder of the fluid was filtered through a graded collodion membrane with an average pore diameter of 0.46 micron, and was then repeatedly instilled into the scarified conjunctiva of the right eye of baboon 1F, all the filtrate being utilized in this way. The monkeys were separately caged throughout the experiments. The unfiltered material yielded a few colonies of Staphylococcus albus and diphtheroid bacilli on culture on plain agar and broth, blood agar and semisolid 10 per cent serum (Leptospira) medium. The filtrate was bacteriologically sterile, and the membrane failed to permit passage of a culture of Bacillus prodigiosus as indicated by tests after filtration of the experimental material.

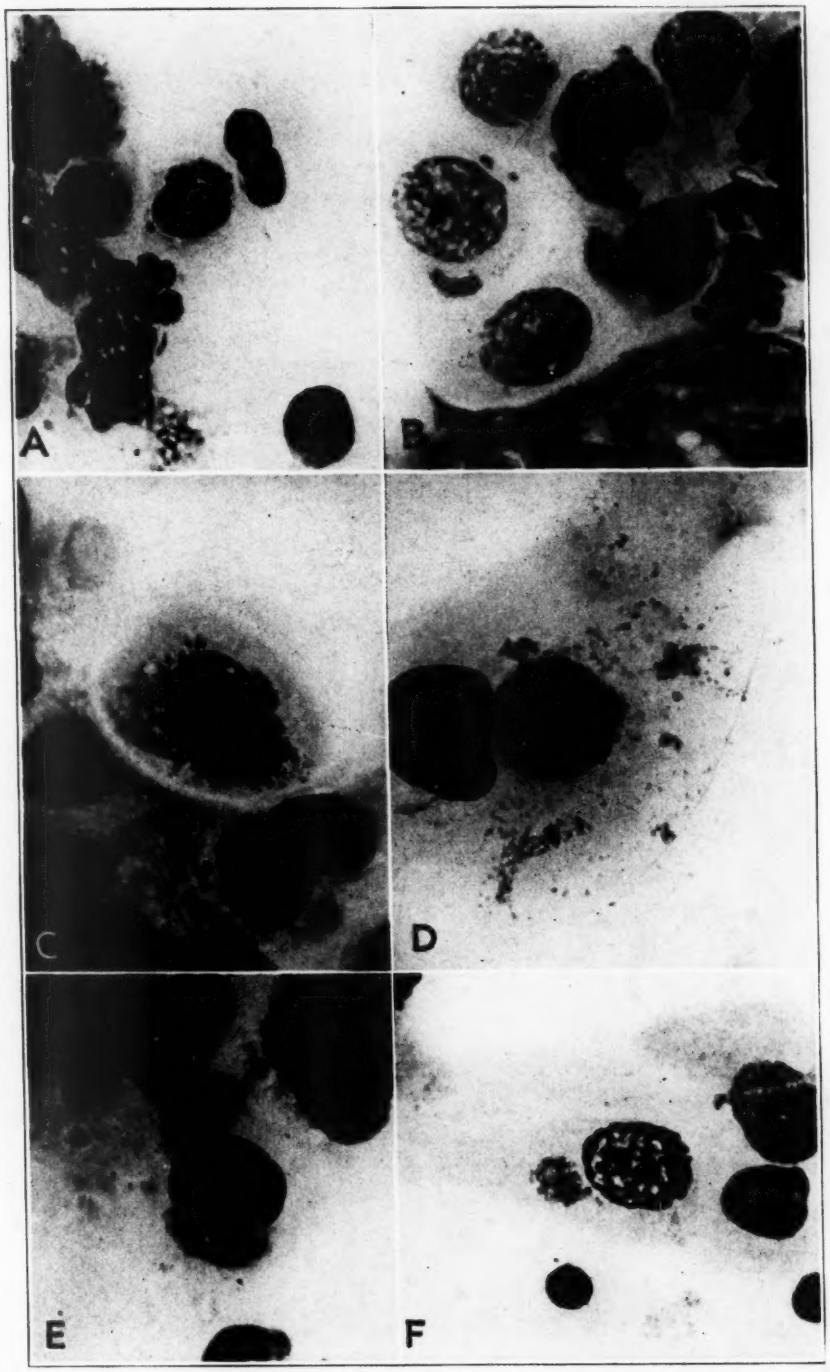
After forty-eight hours, in baboon 1 moderate swelling and redness of both the upper and the lower fold were evident, and in baboon 1F there was slight swelling of the lower fold.

After ninety-six hours, swelling and redness were present in both folds of the inoculated conjunctiva of both animals. The upper tarsus was not affected in either animal.

After one week, the tarsus and the upper fold were clear in both monkeys, and the lower fold showed the same condition as before, the swelling being considerably greater in baboon 1 and the semilunar fold being involved. In both animals there was considerable secretion. The left eye, which had not been inoculated, was not affected in either animal.

After twelve days, diffuse conjunctivitis resembling human inclusion blennorrhea, with involvement of the semilunar fold, was shown on the side of inoculation in both animals. Smears stained with the Giemsa solution showed a number of typical inclusions in the epithelial cells from the eye of baboon 1 (figs. *A* and *B*). No inclusions were observed in the scrapings taken from baboon 1F, and a week later, i. e., at the end of twenty-one days, this animal was definitely recovering, while the condition of baboon 1 remained the same and inclusions were still present in the smears. Twenty-four and thirty-three days after inoculation smears were again made of material obtained from each animal, but no inclusions were noted. Baboon 1F had completely recovered on the twenty-fourth day, but baboon 1 continued to show considerable redness, swelling and mucous secretion.

EXPERIMENT 2.—In each animal the eye which had not been inoculated (the left) remained free from infection. This eye was inoculated at the end of forty days with epithelial scrapings taken from baby W., smears of which (made three days prior to inoculation of the monkey and ten days after birth) contained numerous mixed inclusions (elementary and initial bodies) but no gonococci or other bacteria (figs. *C* and *D*). The preparation of the material and the amounts used were the same in this experiment as in experiment 1, and the method of inoculation was similar, except that 0.2 cc. of the material was injected subconjunctivally and the lids were scarified with the charged needle. The remaining



Photomicrographs of portions of smears prepared from epithelial scrapings. Smears were fixed for fifteen minutes in methyl alcohol, stained two hours in Giemsa's solution (1 drop of the stain to 10 drops of distilled water), decolorized with two changes of 95 per cent alcohol for five seconds each, washed in water, dried in the air and mounted in cedar oil.

A (× 750) shows a mixed inclusion in material obtained from baboon 1 in experiment 1; *B* (× 1,175), several cells from the same monkey, each containing more than one inclusion; *C* (× 1,175), elementary body inclusion, from baby W; *D* (× 1,175), a ruptured cell, with scattering of the elementary bodies, from baby W; *E* (× 1,175), mixed inclusion, from baboon 1F in experiment 2, and *F* (× 1,050), elementary body inclusion, from baboon 1F in experiment 2.

fluid was repeatedly instilled after scarification. Baboon 1F was used for the filtrate, as before, and baboon 1, for the unfiltered material. The average pore diameter of the membrane used in this experiment was somewhat larger (0.62 micron) than that of the membrane employed in experiment 1, but B. prodigiosus failed to pass through the filter, and the filtrate was bacteriologically sterile.

After six days, slight swelling and redness were present in both monkeys.

After sixteen days, definite lesions in both the upper and the lower lid of the left eye were evident in both animals, the tarsus also being slightly reddened. Smears, however, showed no inclusions at this time.

After twenty-five days, severe inflammation was present in both animals, and smears showed typical inclusions in both instances (figs. *E* and *F*).

The condition began to regress about this time, and inclusions were not observed on subsequent examination, though follicles were present and the swelling persisted for four months in both animals.

SUMMARY

The two experiments reported in this paper furnish additional evidence that the virus of inclusion blennorrhea is filtrable through graded colloidal membranes with an average pore diameter of 0.46 to 0.62 micron and is transmissible to the sphinx baboon. The morphologic elements (elementary and initial bodies) observed constantly in the cytoplasm of epithelial cells of the conjunctivae of babies with the acute stage of inclusion conjunctivitis were observed to be associated with the disease in the baboon, whether produced by filtered or by unfiltered material. Although inclusions have been observed in experimental inclusion blennorrhea produced by filtrates in man (Thygeson [1]), they have not been reported in the disease produced by filtrates in baboons. The observation of inclusions in material obtained from baboons reported in this paper add further evidence of their significance in the etiology of the disease.

EPITHELIAL INLAY IN CASES OF REFRACTORY ECTROPION

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Nearly twenty years ago I introduced into plastic surgery a method to transplant free skin flaps of varying size. This method is now generally accepted in reconstructive surgery under the name of epithelial inlay and is highly esteemed not only in Austria and Germany but also in England and in the United States.[1]

This method may also be used in ophthalmic practice when after enucleation the conjunctival socket is not large enough to receive a glass eye, when there is extensive mutilation of the eyelids by fire, when it is necessary to construct new eyelids, etc. I shall now consider its value only in cases of ectropion in which the usual operations may have been unsuccessful. The ectropion may be the result of a burn, lupus or other accident or illness.

The principles of the epithelial inlay are that an exact model is made with Stent's mass, a material used by dentists to get the shape of the jaw. The material becomes soft when placed in hot water and then becomes stiff in a few minutes if placed on the jaws, or, in the case of epithelial inlay, on wounds, where free skin grafts are to be used. The areas may be of any size, as plenty of thin skin is available on both thighs; in case more skin is needed than can be removed from the thighs, it can be taken from anywhere on the body. I have constructed new urinary bladders and esophagi by this method. The model taken of the wound is covered with a skin graft. The thinner the graft, the surer it will heal. The outer side of the skin graft is placed on the model which is replaced on the wound for one week exactly as it was when it was made. The skin is taken from the inner and anterior side of the thigh, or, if only small pieces are wanted, it may be taken from the inner side of the upper portion of the arm, as the skin is very thin in both places. Firm pressure on the wound prevents the accumulation of any secretion between the wound and the graft. With other methods the secretion would separate the graft from the wound entirely or partly. If the technic of the epithelial inlay is carried out correctly, the graft heals completely, and though I have made many hundred epithelial inlays, I have considered the result unusual if the graft had failed to "take" when the Stent mass was removed after a week. The wounds of

1. Reconstructive Surgery, book review, Brit. M. J. **1**:203 (Feb. 2) 1935.

the thigh heal in a few days under a dry dressing or one treated with petrolatum, and afterward, if necessary, other grafts can be taken from the same spot if the grafts are thin. The dressings are left on the thigh until they become displaced of themselves.

REPORT OF A CASE

The following case illustrates my procedure:

A Spanish girl, aged 17, fell into a fire during an epileptic attack and was seriously burned on the face. After the burns healed large scars disfigured the

Fig. 1.—Photograph of patient before grafting of epithelial inlays.

face and neck, and the patient could not lift her head (fig. 1) after the accident. The absence of a lower lip and left lower eyelid caused much annoyance and depressed the patient greatly, and the latter constituted a danger to the eye. In addition there was an ectropion of the right lower lid. A surgeon had tried in vain to make a new left lower lid by using two large pedicled skin flaps, one from the forehead above the right eyebrow and one from the cheek in front of the left ear. These flaps were sewed together, but the result was nil, and the disfigurement was much increased. The right eyebrow was displaced nearly 2 cm. above the left, and there were disfiguring scars on the forehead and especially on the cheek. The ectropion was maximal, and there was practically no lower eyelid.

Professor Marquez, the president of the faculty of medicine of the University of Madrid, sent this patient to me, and I operated on her on March 8, 1935, in the surgical clinic of Professor Estella, University of Madrid.

I made four epithelial inlays at one time: one large one for the neck (a whole thickness graft), another to make a new lower lip and one for each ectropion. All four healed.

I shall not speak here of the first two but shall limit my remarks to the last two, as they constituted ophthalmologic problems. First of all, I made a deep cut along the eyelid, nearly at right angles to the surface and somewhat inclined, in order to separate the deep lying conjunctiva and the muscles close to it, which were not cicatricial, from the greater part of the muscles of the lid, which grew more and more scarlike the nearer they approached the skin of the cheek. Then I performed a tarsorraphy on each eye and fastened the sutures to the skin above

Fig. 2.—Photographs of patient three weeks after grafting of epithelial inlays.

the eyebrows in order to raise the palpebral fissures as much as possible and to spread the margins of the wound.

Models in Stent's mass were made of both large wounds, and skin grafts from the thigh were placed on the part of each model which was to come in contact with the wound. Then the molds were placed on the wounds, and sutures of strong silk were applied tightly in U shape over the molds from the forehead to the cheek to press the molds against the wounded surface. After a week the sutures were removed. The results three weeks later are shown in in figure 2. At a later date I shall undertake further reparative procedures.

BIOCHEMISTRY OF THE LENS

VII. SOME STUDIES ON VITAMIN C AND THE LENS

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The lens, being devoid of blood vessels in postnatal life, is dependent on its surrounding media for nutrition and respiration. However, because of its internal oxidative mechanism, it is partly independent of the aqueous for its respiration. It is fortunate that this is so, for, according to Duke-Elder,[1] the oxygen tension of the aqueous is insufficient to meet the requirements of the lens. The internal oxidative system of the lens functions through the agencies of glutathione and vitamin C, or cevitamic acid. The latter is present in the aqueous and lens, while the former is found only in the lens.

These substances are capable of being reversibly oxidized and reduced, thus forming a so-called "redox" system. This property depends on the ability of glutathione and vitamin C to accept hydrogen, and in this process the hydrogen donor (the lens) is oxidized. That the reversibly oxidized vitamin C is reduced by the lens appears probable from the report of Buschke and Goldmann.[2] These writers found that vitamin C appears in the reduced form only in the normal aqueous, whereas in the aphakic eye the vitamin C content of the aqueous drops markedly, and that which is present is in the reversibly oxidized form. Vitamin C, like glutathione, is found to be diminished in quantity or absent in the cataractous lens (von Euler and Martius).[3]

The question whether the absence of cevitamic acid in the cataractous lens precedes the opacification or is merely a result of this condition cannot be answered definitely at present. Some experiments, however, point to the former possibility. Monjukowa and Fradkin[4] were able to produce cataract in a small percentage of guinea-pigs by feeding them a scorbutic diet, and if the aqueous was drained by paracentesis cataract

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1. Duke-Elder, W. Stewart: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1934, vol. 1, p. 479.

2. Goldmann, H., and Buschke, W.: Klin. Wchnschr. **14**:239, 1935.

3. von Euler, Hans, and Martius, Carl: Ztschr. f. physiol. Chem. **222**:65, 1933.

4. Monjukowa, N. K., and Fradkin, M. J.: Arch. f. Ophth. **133**:328, 1935.

developed in all of the animals. Bietti and Carteni [5] found that the quantity of cevitamic acid in the crystalline lens fell to a mere trace in guinea-pigs on a diet deficient in vitamin C. I [6] found that there was a tendency to a low vitamin C level in the blood of cataractous subjects. These facts point to the possibility that a diminution in the vitamin C content of the eye, whether due to a deficiency in the diet or to some barrier in the blood vessels, may result in a cataract. In view of the importance of vitamin C in the physiology of the eye, it was thought that some further experiments with this substance were justified.

METHOD OF DETERMINATION

The method used for the determination of cevitamic acid and glutathione in the ocular tissues consisted of a combination of several procedures. A 2 per cent solution of meta-phosphoric acid was used in the method recommended by Fujita and Iwatake [7] for the extraction of cevitamic acid and glutathione. The total iodine-reducing substances present were determined by the method of Woodward and Fry,[8] a procedure which they used for the determination of the glutathione in the blood. The amount of cevitamic acid was estimated by the method of Birch, Harris and Ray [9] in which sodium 2,6 di-chlorobenzenone-indophenol is used as an indicator. One obtains the quantity of glutathione by subtracting the amount of iodine reduced by the cevitamic acid from the total iodine-reducing substances present.

PROCEDURE

A lens was ground up in a mortar with a fresh 2.5 per cent solution of meta-phosphoric acid (10 cc.) and a little sand. Meta-phosphoric acid was used instead of sulfosalicylic acid because the latter assumes a pinkish color (probably due to iron present in the distilled water) which interferes in determining the end-point for vitamin C. The extraction was carried out twice. The combined liquid was centrifugated. An aliquot portion of the supernatant fluid was placed in a flask; to this were added 2 cc. of a fresh 5 per cent solution of potassium iodide, 2 cc. of a 5 per cent solution of meta-phosphoric acid and 1 drop of a 1 per cent solution of starch. This was titrated against thousandth-normal potassium iodate made up in a 2 per cent solution of sulfosalicylic acid to maintain the desired acidity. The solution was standardized against glutathione, which has a melting point of 190 C. It was found that 3.145 cc. of thousandth-normal potassium iodate was necessary for 1 mg. of pure glutathione. The amount of potassium iodate obtained thus represented the amount of reducing substances present (glutathione and vitamin C).

An aliquot portion of the supernatant fluid was titrated against the standardized dye (sodium 2,6 di-chlorobenzenone-indophenol). The value obtained is the

5. Bietti, G., and Carteni, A.: Boll. d. Soc. ital. di biol. sper. **9**:983, 1934; abstr., Nutrition Abstr. & Rev. **5**:376, 1935.

6. Bellows, J.: Biochemistry of the Lens: V. Cevitamic Content of the Blood and Urine of Subjects with Senile Cataract, Arch. Ophth. **15**:78 (Jan.) 1936.

7. Fujita, A., and Iwatake, D.: Biochem. Ztschr. **277**:293, 1935.

8. Woodward, G. E., and Fry, E. G.: J. Biol. Chem. **97**:465, 1932.

9. Birch, T. W.; Harris, L. J., and Ray, S. N.: Biochem. J. **27**:590, 1933.

amount of vitamin C present. By changing this value into terms of thousandth-normal potassium iodate and subtracting this amount from the total quantity of potassium iodate titrated, one obtains the amount reduced by glutathione.

That the method used for the determination was accurate was shown by tests for recovery (table 1). In these tests known amounts of cevitamic acid and glutathione were added to lens extract which had previously been examined for these substances. The amount of recovered vitamin C was 100 per cent, and that of glutathione, 95 per cent.

EXPERIMENT 1.—The glutathione and vitamin C contents of a mature and an immature senile cataract from a human being were determined. The mature senile cataract, weighing 117 mg., did not contain an appreciable amount of glutathione or vitamin C, and the immature senile cataract (brown), weighing 228 mg., contained 81.9 mg. of glutathione per hundred grams and 5.7 mg. of cevitamic acid. According to the figures of Müller and Buschke, the normal lens of a human being contains 29.6 mg. of cevitamic acid per hundred grams and a bovine lens contains 129.7 mg. of glutathione and 32.7 mg. of cevitamic acid.

The figures given here are in accordance with those of other workers. The amounts of glutathione and vitamin C were diminished in the immature cataract, while in the mature cataract these substances were not present.

TABLE 1.—*Results of Tests for the Recovery of Glutathione and Cevitamic Acid*

	Amount Present in Lens Extract, Mg.	Amount Added, Mg.	Amount Recovered, Mg.
Glutathione	0.081	0.020	0.100 (95%)
Vitamin C	0.005	0.012	0.017 (100%)

EXPERIMENT 2.—The absorption of cevitamic acid from the conjunctival sac of a rabbit was demonstrated. Powdered vitamin C [10] was placed in the conjunctival sac of a rabbit, and the closed lids were massaged over this. (A total of 100 mg. was used.) Twenty hours later this procedure was repeated with 50 mg. of the powder. After one hour the conjunctival sac was washed with a solution of boric acid. Following this, under butyn anesthesia, a fine needle at the end of a syringe was introduced into the anterior chamber, and a few drops of aqueous humor was removed. The chamber was not completely emptied, because it was thought that the last of the aqueous would contain some second aqueous, which has been shown to contain little vitamin C. The aqueous of the fellow eye was used as a control, because it was shown by Müller and Buschke [11] that the concentration of this substance in either eye of an animal is the same. The aqueous was immediately mixed with a 2 per cent solution of meta-phosphoric acid (fresh) and centrifugated. Aliquot portions of the supernatant fluid were titrated for vitamin C concentration by means of the dye in a microburet graduated in 0.002 cc. The concentration in the treated eye was greater than in the control (table 2).

The values are probably low because the small amounts of aqueous obtained suffered partial oxidation during the weighing process, but the relative values would be unchanged. These experiments prove that

10. Cebione-Merck was used.

11. Müller, H. K., and Buschke, W.: Arch. f. Augenh. **108**:592, 1934.

vitamin C is absorbed from the conjunctival sac into the aqueous humor. From the work done on alkaloids, it is known that this must take place through the cornea. If it can ever be shown that an artificial increase of the amount of vitamin C in the lens is of value in arresting the progress of a senile cataract, this experiment would indicate a means by which such increase can be effected, for by this method any vascular barrier to the passage of vitamin C from the blood into the anterior chamber is overcome. Such a condition is conceivable in cases of vascular sclerosis or of the inflammatory conditions leading to cataracta complicata. However, no satisfactory evidence has been reported that would indicate an effect of vitamin C on the progress of cataract.

The Effect of the Lens on Vitamin C.—It is well known that vitamin C in a neutral or alkaline solution is readily oxidized in air. Müller and Buschke [11] demonstrated that aqueous humor containing a lens in vitro shows little diminution in its vitamin C content, while a similar amount

TABLE 2.—*Concentration of Vitamin C in the Aqueous of a Treated and of a Control Eye*

	Amount of Dye Reduced, Cc.	
	1st Reading	2d Reading
Rabbit 1*		
Aqueous from the eye treated with vitamin C	0.008	0.010
Aqueous from the control eye	0.005	0.004
	Vitamin C in Mg. per 100 Gm.	
Rabbit 2		
Aqueous from the eye treated with vitamin C	23	
Aqueous from the control eye	5	

* Prof. C. A. Farmer checked my results in this case.

of aqueous without a lens suffers a marked loss of this substance within a few hours. Green [12] explained the preservation of vitamin C by the presence of an antioxidative substance in the tissues. The effect of the lens and of other substances on vitamin C is demonstrated in the following experiments.

EXPERIMENT 3.—Equal quantities of vitamin C were placed in two test tubes and to each 5 cc. of a phosphate buffer solution of p_H 7.4 was added. The lens of a guinea-pig was placed in one tube. The tubes were placed in an incubator at 37 C. The amounts of vitamin C recovered after two and a half and seventeen hours are given in table 3.

Again, equal quantities of vitamin C were placed in test tubes. To each was added 5 cc. of a phosphate buffer solution of p_H 7.4. A crystalline lens of a dog was added to one test tube, a small bead of glutathione to another, and the ash of a crystalline lens of a pig to another, and the final tube was used as a control

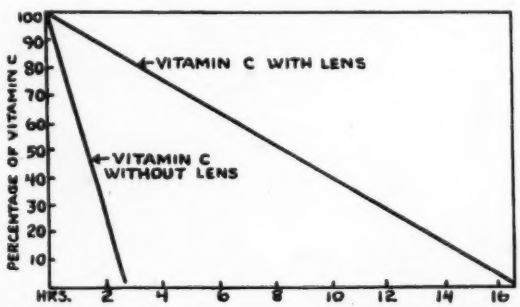
12. Green, D. E.: Biochem. J. **27**:1044, 1933.

The whole series was placed in an incubator at 37 C. for one hour. The effect of these various substances on the amount of vitamin C is shown in table 4.

One sees from the foregoing experiments that vitamin C is preserved by the presence of crystalline lens or glutathione. The vitamin C does not escape from the lens into the solution to cause this difference. This is shown by a comparison of the amounts of this substance in the lens used in the solution and that of its fellow lens, the difference in this

TABLE 3.—*Results Showing the Preservative Effect of the Lens on Vitamin C*

	Amount of Vitamin C Added, Mg.	Amount of Vitamin C Recovered, Mg.	
		After 2½ Hr.	After 17 Hr.
Solution of guinea-pig lens	0.098	0.087 (89%)	0.011 (11%)
Control	0.098	0.003 (3%)	0



Graphic presentation of the preserving action of the guinea-pig lens on vitamin C.

TABLE 4.—*Effect of Various Substances on Vitamin C*

Substance Added	Amount of Vitamin C Added, Mg.	Amount of Vitamin C at End of 1 Hr., Mg.
Lens	0.098	0.018 (18%)
Control	0.098	0.005 (5%)
Glutathione	0.098	0.031 (32%)
Ash of lens	0.098	0.008 (8%)

Amount of cevitamic acid found in the first lens.....0.028 mg.
Amount of cevitamic acid found in the fellow lens.....0.032 mg.

case being only 0.004 mg. Because of the marked preserving action of glutathione, one might be led to believe that the preserving action of the lens depends on the presence of this substance. In order to determine whether or not the glutathione in the lens is the substance which preserves the vitamin C, the following experiment was undertaken.

EXPERIMENT 4.—A cataractous lens was extracted with a 2.5 per cent solution of meta-phosphoric acid. This extract gave no appreciable yield in reducing sub-

stances (glutathione or vitamin C) when titrated against thousandth-normal potassium iodate in a fine capillary microburet. Likewise, an immature cataractous lens was extracted which yielded 0.188 mg. of glutathione and 0.013 mg. of vitamin C. Of three test tubes, each containing 0.136 mg. of vitamin C, an aliquot portion of the extract of the first lens was added to one and an equal amount of the second lens extract to the second and the third was the control. A phosphate buffer solution of p_H 7.4 was added, and the whole was incubated at 37 C. The result showed that the preserving action of the mature cataract without glutathione was equal to that of the immature lens with a relatively large quantity of glutathione. Furthermore, the extract of a lens is quite as effective in preserving vitamin C as small pieces of tissues from other organs but is less effective than whole lens material.

TABLE 5.—*Preserving Action of Various Lenses on Vitamin C*

Animal	Amount of Vitamin C Added, Mg.	Amount of Vitamin C Recovered	
		After 1 Hr., Mg.	After 2 Hr., %
Dog	0.098	0.018	18
Dog	0.081	0.073	90
Rat	0.098	0.077	79
Guinea-pig	0.098	0.090	98
Rabbit	0.081	0.089	103*

* Experimental error up to 5 per cent may explain this, yet the possibility exists that some vitamin C may be formed in the lens.

TABLE 6.—*A Comparison of the Preserving Action of Various Organs on Vitamin C*

	Amount of Vitamin C Added, Mg.	Amount of Vitamin C Recovered			
		After 1 Hr.		After 2 Hr.	
		Mg.	%	Mg.	%
Guinea-pig					
Lens	0.040	0.039	98	0.033	83
Liver	0.040	0.004	10	0.003	8
Spleen	0.040	0.004	10	0.003	8
Adrenal	0.040	0.008	20	0.006	15
Heart muscle	0.040	0.004	10	0.004	10
Control	0.040	0.004	10	0.002	5
Rat					
Lens	0.082	0.081	99	0.070	85
Liver	0.082	0.075	91	0.062	76
Spleen	0.082	0.039	48	0.031	37
Adrenal	0.082	0.013	16	0.013	16
Heart muscle	0 082	0.013	16	0.010	12
Control	0.082	0.008	10	0	0

From these experiments it is seen that although glutathione itself has a marked preserving action on vitamin C, a cataractous lens with no demonstrable glutathione is as effective as one with glutathione. This confirms Mawson's [13] report that dialyzed tissues containing no glutathione were found to be effective in preserving vitamin C in alkaline solutions. The preserving action of various lenses on vitamin C is shown in table 5.

13. Mawson, C. A.: Biochem. J. **29**:569, 1935.

In spite of the great differences in the size of the lens in the animals used in the experiments recorded in table 5, the preserving action is very similar and within certain limits seems to be independent of the amount of lens.

EXPERIMENT 5.—A comparison was made of the preserving action on vitamin C of equal weights of various organs of an animal in a phosphate buffer solution of p_H 7.4 at 37 C. (table 6).

One sees from this table that the lens exceeds by far the other organs in preserving vitamin C in the reduced form.

CONCLUSION

The remarkable preservative action of the lens on vitamin C as compared to other tissues seems to indicate its importance for maintaining the proper respiratory mechanism of the lens on which it is dependent for life. An interesting related fact is that the lens is also richer in glutathione and vitamin C than the other organs of the body. It would seem from the experimental work reported here that the antioxidative action of the lens on vitamin C does not depend on its content of glutathione and that still another factor must be present in the lens tissue which is important in preserving the respiratory mechanism of the lens. Although this work showed only the preservative action of vitamin C in the aqueous, it must be considered that such an effect takes place in the lens itself. This is borne out by the small loss of vitamin C sustained by the lens in the alkaline solution for one hour (experiment 4).

This action of the lens offers an explanation of the absence of the reduced form in the aqueous of the aphakic eye and the presence of only small amounts of the reversibly oxidized form of the vitamin C. With the removal of the lens, the mechanism for preventing the oxidation of the vitamin C in the aqueous which is a slightly alkaline solution (p_H 7.35) is lost. This explanation seems more plausible than that of Müller and Buschke [14] and of Fischer,[15] who explained this finding on the basis that the lens itself forms the vitamin C.

14. Müller, H. K., and Buschke, W.: Arch. f. Augenh. **108**:368, 1934.
15. Fischer, F. P.: Klin. Wchnschr. **13**:596, 1934.

CIRCULATION OF THE AQUEOUS

V. MECHANISM OF SCHLEMM'S CANAL

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In previous papers Dr. Pierce and I [1] have reported the experimental results in a systematic study regarding the mode of reabsorption of the various components of the intra-ocular fluid. These results may be summarized as follows: The reabsorption of water, of crystalloids and of colloids from the anterior chamber is in each instance effected by a separate and essentially unrelated mechanism. Colloids are removed by active phagocytosis on the part of the anterior layer of the cells of the iris, the endothelium. The removal of proteins is facilitated by the presence of proteolytic enzymes in the aqueous and the surrounding tissues. Crystalloid exchange takes place between the anterior chamber and the blood vessels of the iris by diffusion. The velocity of approach toward equilibrium in crystalloid content between the blood and the aqueous is controlled by the area of the iris (i. e., the state of contraction or dilatation of the pupil) and by the degree of hyperemia of the iris. In smaller part, some of the crystalloids of the aqueous are carried out of the anterior chamber along with the reabsorbed water.

In regard to the reabsorption of water we have shown, first, that this reabsorption takes place practically exclusively from the anterior chamber. Only the vessels of the iris and Schlemm's canal need, therefore, be considered as possibly concerned in this mechanism. Secondly, we showed that congestion of the anterior ocular vessels, whether active or passive, tended to facilitate, within limits, the reabsorption of water. It is evident that with a rise in the pressure within the vessels of the iris as a result of such congestion fluid should tend to enter the anterior chamber rather than to be withdrawn from it. From this we concluded that the iris plays only a subsidiary rôle in the intra-ocular fluid exchange. We were thus led, by a process of exclusion, to the conclusion that the chief mechanism concerned in the reabsorption of water from the eye is Schlemm's canal, and have suggested a possible mode of operation of the canal which would explain the seemingly para-

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From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Friedenwald, J. S., and Pierce, H. F.: Circulation of the Aqueous: II. Mechanism of Reabsorption of Fluid, Arch. Ophth. **8**:9 (July) 1932; III. Reabsorption of Crystalloids, ibid. **10**:449 (Oct.) 1933; IV. Reabsorption of Colloids, ibid. **14**:599 (Oct.) 1935.

doxical fact that congestion of the anterior ocular vessels causes an increase in the rate of reabsorption of water. In the present paper it is proposed to formulate this hypothesis in greater detail and to present evidence which substantiates it.

The experiments of Duke-Elder [2] and those that we have reported indicate that the hydrostatic pressure within Schlemm's canal is equal to or slightly above that within the anterior chamber. It is impossible, therefore, to assume as has been done in the past that fluid enters Schlemm's canal by simple hydrostatic filtration. On the contrary, fluid can be drawn into the canal only by one or the other of two possible mechanisms: Either there is an active secretion of fluid into the lumen of the canal by its endothelial wall, or plasma proteins must be present within the canal in sufficient concentration to attract water osmotically from the anterior chamber into the canal. There is no evidence to indicate that a single layer of endothelium is capable of active secretion, and certainly such a vitalistic theory could not reasonably be suggested until the simpler physical possibilities have been excluded.

Since the hydrostatic pressure within the canal cannot far exceed that in the anterior chamber the concentration of plasma proteins required for the effective operation of the canal needs be quite small. Duke-Elder, for instance, estimates the pressure within the canal to be only 2 or 3 mm. of mercury higher than that in the anterior chamber. On the other hand, the osmotic pressure of the plasma proteins at their normal concentration is from 25 to 30 mm. of mercury. It follows that at equilibrium between the canal and the anterior chamber the plasma would be diluted about tenfold, and any increase in concentration of the plasma over this would entail the osmotic attraction of water from the anterior chamber into the canal.

It is evident that the hypothesis which we have formulated requires that plasma be supplied continuously to the canal from the blood vessels; that is to say, there is a constant stream of plasma into the canal from afferent arterioles just as there is a constant stream of plasma plus a greatly increased volume of water leaving the canal by efferent venules. Our conception, therefore, is that the canal lies between artery and vein as a specialized form of capillary sustaining a slight but steady through and through circulation of blood or plasma, rather than a blind outpouching of the episcleral veins as has hitherto been assumed.

The existence of afferent arteriolar connections with Schlemm's canal is a matter that may readily be demonstrated by histologic study, and it can be admitted at once that such afferent vessels are easily found. Indeed, it is somewhat surprising that they have not been discovered

2. Duke-Elder, W. S.: Brit. J. Ophth. **10**:513, 1926.

hitherto. Thus Maggiore [3] in his classic studies on the anatomy of the canal describes only efferent connections, but his studies were based on specimens in which he had filled the canal with an injection mass introduced via the carotid arteries. It must be obvious that if the canal were merely a blind outpouching of the veins, as Maggiore concluded, one could not readily fill it by intravascular injection, for where could the normal fluid content run out? I have repeated Maggiore's experiments on rabbits and have found little difficulty in filling the canal with an injection mass introduced through the carotid arteries.

Sondermann [4] in his studies on the anatomy of Schlemm's canal was chiefly concerned with problems of embryology. He showed that the anlage of the canal is a part of the iridociliary and intrascleral vascular net. Convinced that Schlemm's canal is a venous sinus, he concluded that the vessels afferent to and efferent from this vascular net are veins but furnished no proof of this statement.

Theobald [5] in her beautiful reconstruction of Schlemm's canal confirmed the general anatomic findings of Maggiore and Sondermann and concluded: "There are no afferent vessels to the canal." But in the legend for her illustration, model 2-B, she said, "Note: The arteriole becoming a venule, and later receiving the upper collector from the canal of Schlemm." It should be clear, however, that the mere fact that a branch or continuation of an artery happens to be devoid of a muscular coat does not justify the assumption that the flow within that vessel normally goes toward the artery. It follows that the facts presented by Theobald do not warrant the categorical conclusion that vessels afferent to the canal are wholly lacking.

The data to be presented here are based on the study of serial sections of the anterior segments of four eyes, all essentially normal in regard to the portions under consideration. One of the four eyes was embedded in pyroxylin (celloidin) and cut at 15 microns. The remaining three were embedded in paraffin and cut at 7 microns. Since, with the important exceptions to be specially noted, the results of this study agree with those of Theobald and Maggiore, they will be presented in summary only. In all four eyes the canal was found to have three types of connections: (1) efferent venules, (2) afferent arterioles and (3) so-called inner canals.

THE EFFERENT VENULES

The observations in regard to the efferent venules are entirely in accord with those reported by Theobald. These vessels, upwards of twenty in number, join the canal with the intrascleral venous plexus

3. Maggiore: Ann. di ottal e clin. ocul. **40**:317, 1917.
4. Sondermann: Arch. f. Ophth. **124**:521, 1930; **126**:341, 1931.
5. Theobald, Georgiana Dvorak: Tr. Am. Ophth. Soc. **32**:593, 1934.

which lies about midway in the thickness of the corneoscleral tissues at the limbus. They are lined by endothelium which lies directly on the fibers of the corneal stroma without intervening adventitia. Most often they are greatly flattened so that a cross-section may suggest the appearance of an oblique longitudinal section, and their dimensions can be accurately seen only when serial sections are studied. They reach their destination in the intrascleral venous plexus by a course which is sometimes straight but often rather tortuous. Occasionally they send a continuing branch to the episcleral vessels at the limbus.

THE AFFERENT ARTERIOLES

The afferent arterioles are much less numerous than the efferent venules and have hitherto apparently completely escaped notice. They occur as branches of the arterioles supplying the intrascleral plexus, but not every one of these arterioles possesses such a branch. These arterioles have been well described by both Maggiore and Theobald and are particularly well illustrated in the latter's paper. They arise from the ciliary arteries, enter the inner surface of the sclera at from 0.5 to 1 mm. behind the scleral spur and proceed obliquely forward and outward toward the intrascleral plexus at the limbus. Almost always each is accompanied by a small nerve and this, as well as the obvious thickness of the wall of the arteriole, serves to identify it. The number of these arterioles is about equal to that of the efferent venules, and most of them are not connected with Schlemm's canal. In each eye that I have examined there were, however, approximately from six to eight of these vessels that were connected with the canal, each by a short direct branch. The connection most often takes the form shown in figures 1 and 2. The arteriole is seen passing anterior to the canal and separated from it by about one third of the thickness of the cornea. Without deviating appreciably from its course it gives off a branch which, characteristically, forms a slightly acute angle in a retrograde direction with the parent stem and runs directly into the canal. Usually this connecting branch is devoid of any muscular coat and may therefore be readily confused with the efferent venule, but in some instances the wall of the vessel is appreciably thicker than that of the intrascleral venule and a minute twig of the nerve which accompanies the parent arteriole may sometimes be seen to branch off along with the connecting vessel.

These afferent arteriolar branches are usually circular in cross-section and have an internal diameter of only from 5 to 10 microns. The arteriole from which they branch is about one and a half to two times as large while the efferent venules, at least in their larger diameter, usually measure over 20 microns. The small diameter of these afferent

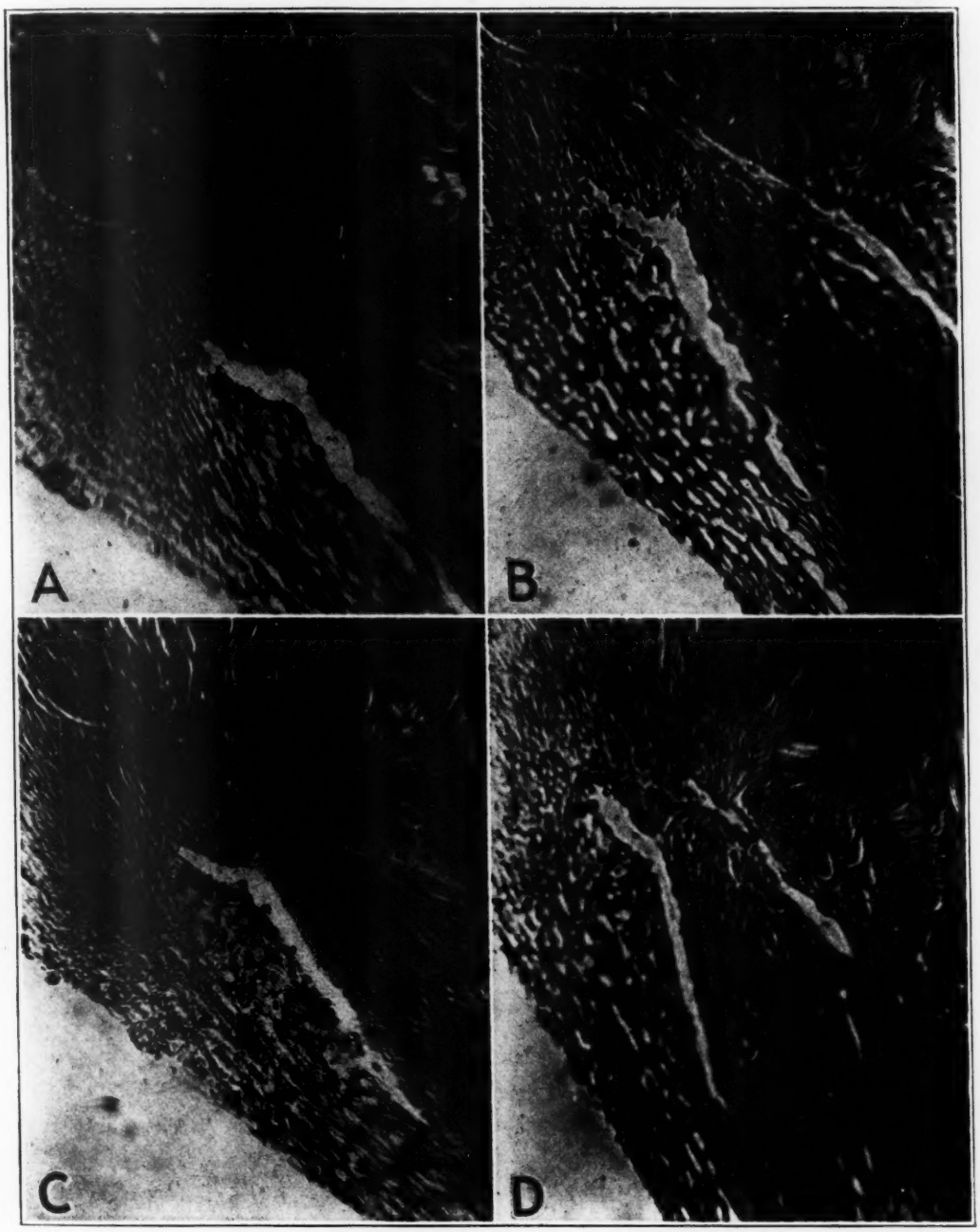

Fig. 1.—Successive serial sections through a vessel afferent to Schlemm's canal.

vessels, together with the slightly retrograde direction of their branching, helps to explain why red blood cells find their way into the canal in such small numbers. Neither of these facts, however, should interfere with the slow trickle of plasma from the arteriole into the canal. In fact, one may readily calculate that if the pressure in the arteriole is only from 5 to 10 mm. of mercury above that in the canal the amount of plasma flow required to keep up the normal rate of circulation of the intra-ocular fluid would be abundantly supplied. In their size, their number and their mode of connection with the canal these afferent vessels satisfy every requirement for the effective operation of Schlemm's canal according to the hypothesis outlined.

Fig. 2.—Schematic representation of the afferent and efferent connections of Schlemm's canal.

THE SO-CALLED INNER CANALS

Sondermann was the first to describe the outpouchings of Schlemm's canal between the trabeculae of the pectinate ligament. Both Sondermann and Theobald believe, on the basis of their histologic observations, that these outpouchings represent open connections between the canal and the anterior chamber, and they have, therefore, named these structures "inner canals." They have thus reopened the ancient controversy between Leber and Schwalbe on this point. It is to be remembered that the classic experiments of these two investigators and their many pupils, carried out with the most meticulous care, finally led to the conclusion

that there are no open connections between the canal and the anterior chamber. The results of these experiments are not lightly to be put aside. The walls of the so-called inner canals when visible consist of a single layer of very much flattened endothelium, so delicate as to appear threadlike even under high power microscopy. That these delicate walls could easily be broken and torn in the process of sectioning can hardly be doubted, and the attempt to follow in serial section the minute spaces which these walls enclose between the fibers of the pectinate ligament is open to grave error. The patency of these openings in life must, therefore, be seriously doubted, and Sondermann's own embryologic studies, which show that the canal embryologically is a plexus of completely walled vascular tubes, furnishes further doubt as to the existence of holes in these walls in the adult. If further support for the conclusions of Leber is required it can be found in the work of Maggiore. The latter, as noted earlier in this paper, found no difficulty in filling the canal with an opaque material injected intra-arterially, but in no case did he find that this injected material ran out of the canal into the anterior chamber. My own experience with Maggiore's technic is similar. The injected mass fills the canal fully but does not leak into the anterior chamber even if the pressure within the eye is reduced by corneal puncture. Finally, the existence of afferent arteriolar connections with the canal precludes the possibility of open connections between the canal and the anterior chamber, for if there were open connections plasma would necessarily leak from the canal into the anterior chamber and raise the protein concentration there. It must be concluded, therefore, that the so-called inner canals are actually blind pockets extending from the canal into the adjacent spaces of the pectinate ligament but separated from the anterior chamber in life by a continuous endothelial wall.

THE PRESENCE OF BLOOD IN SCHLEMM'S CANAL

The conclusions which we have reached cast some new light on the long debated problem of whether or not the canal of Schlemm normally contains blood. Fuchs [6] and Salzmann [7] reported that they had observed the canal to be filled with blood in the living eye. Troncoso,[8] on the other hand, found the canal when observed with the gonioscope to be pale and almost colorless. It is generally admitted that when the eye is congested the canal does in fact contain blood, which may even be demonstrable in histologic sections, though in sections of normal eyes red blood corpuscles are rarely seen within the canal. It should be

6. Fuchs, E.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **28**:136, 1900.
7. Salzmann: Ztschr. f. Augenh. **31**:1, 1914; **34**:26, 1915.
8. Troncoso, M. U.: Am. J. Ophth. **8**:433, 1925.

remembered, however, that when an eye is removed for histologic study the pressure in the vessels connected with Schlemm's canal necessarily falls promptly to zero, and that any additional fluid which may filter into the canal under the influence of the still appreciable intra-ocular pressure must tend to flush out the canal and clear it of the few red blood corpuscles which it might have contained during life. Even if the eye is placed as rapidly as possible in a fixing solution, such filtration should continue for some time because time is required for the fixative to penetrate through the sclera to the canal. A much less efficient mechanism of a similar kind is at hand to empty the retinal arteries of their blood, and one does, in fact, often find them practically empty in histologic sections, but one does not, on that account, conclude that they are empty of blood during life.

From the point of view of the present investigation it is clear that the blood normally entering the canal would be greatly diluted with aqueous, and that the corpuscular content may be expected to be very small if a skimming of the plasma should take place at the origin of the afferent arteriolar branch. It is not surprising, therefore, that the region of the canal when viewed with the gonioscope is practically devoid of color.

OBSERVATIONS WITH POSSIBLE BEARING ON THE THEORY OF GLAUCOMA

In conclusion one may consider what possible bearings the present conception of the mode of operation of Schlemm's canal may have on the theory of glaucoma. It has been shown that hyperemia of the vascular bed with which the canal is connected increases the rate of reabsorption of the intra-ocular fluid. One may conclude that a decrease in the flow of plasma through the afferent arterioles into the canal would decrease the efficiency of the canal in removing fluid from the eye and lead to a rise in the intra-ocular tension. It would be of interest, therefore, to know whether sclerotic changes are to be found frequently in the afferent arterioles of eyes affected with primary simple glaucoma. Particularly it would be important to know whether such changes are found early in eyes with primary simple glaucoma.

The first question can be readily answered in the affirmative. In eyes showing advanced chronic glaucoma, which form the great bulk of those available for histologic study, the afferent arterioles and the arterioles of the intrascleral plexus of which they are branches commonly show marked hyaline degeneration of their walls with reduction in their lumens and even occasional thrombotic occlusion. A few of the changes found are illustrated in figures 3 and 4. But one cannot immediately assume that these arteriolar changes are primary to the glaucoma.

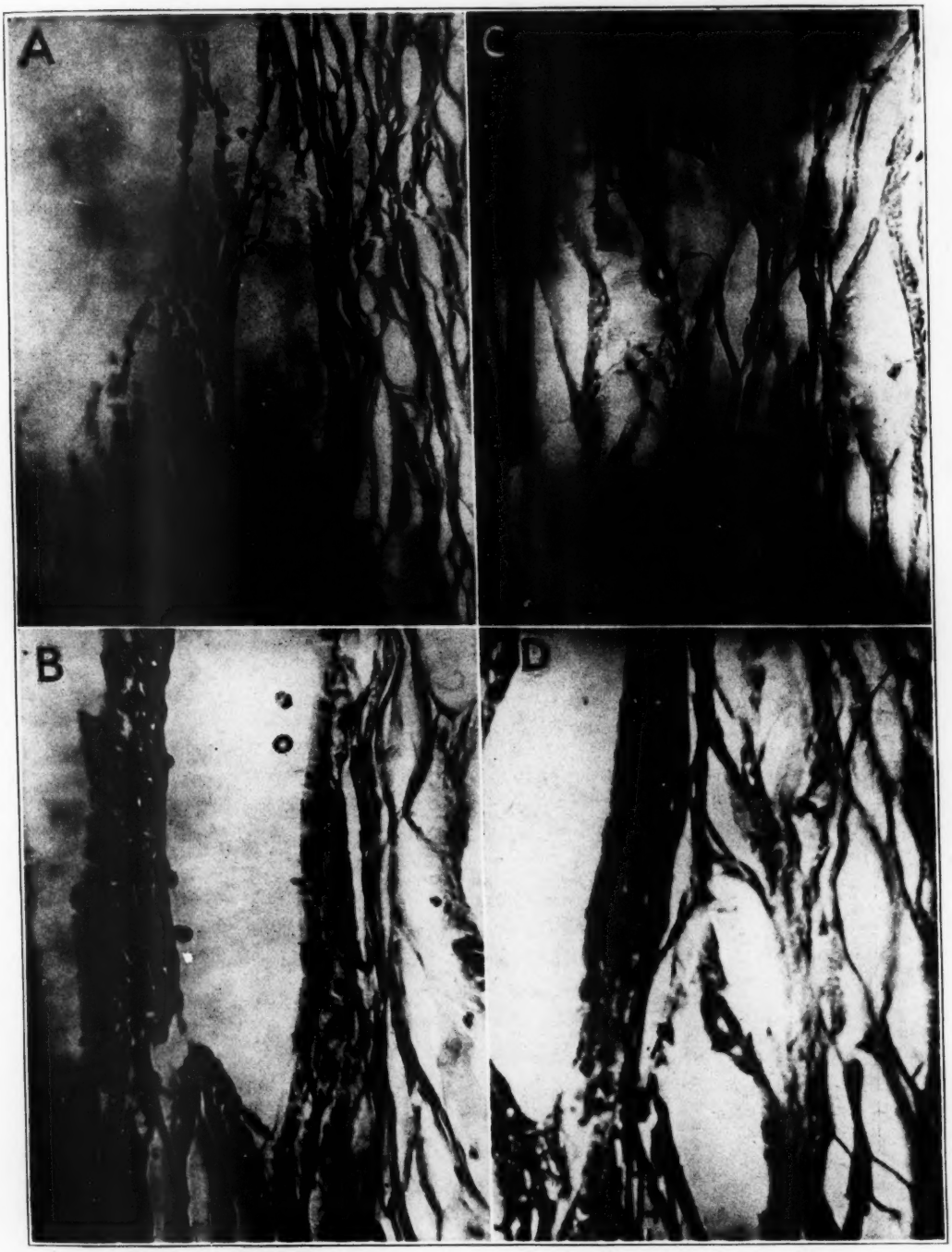

Fig. 3.—Sections from a case of early glaucoma simplex: *A*, Schlemm's canal with normal pectinate ligament; *B*, Schlemm's canal with normal "inner canal"; *C*, and *D*, hyaline degeneration of arterioles afferent to Schlemm's canal.

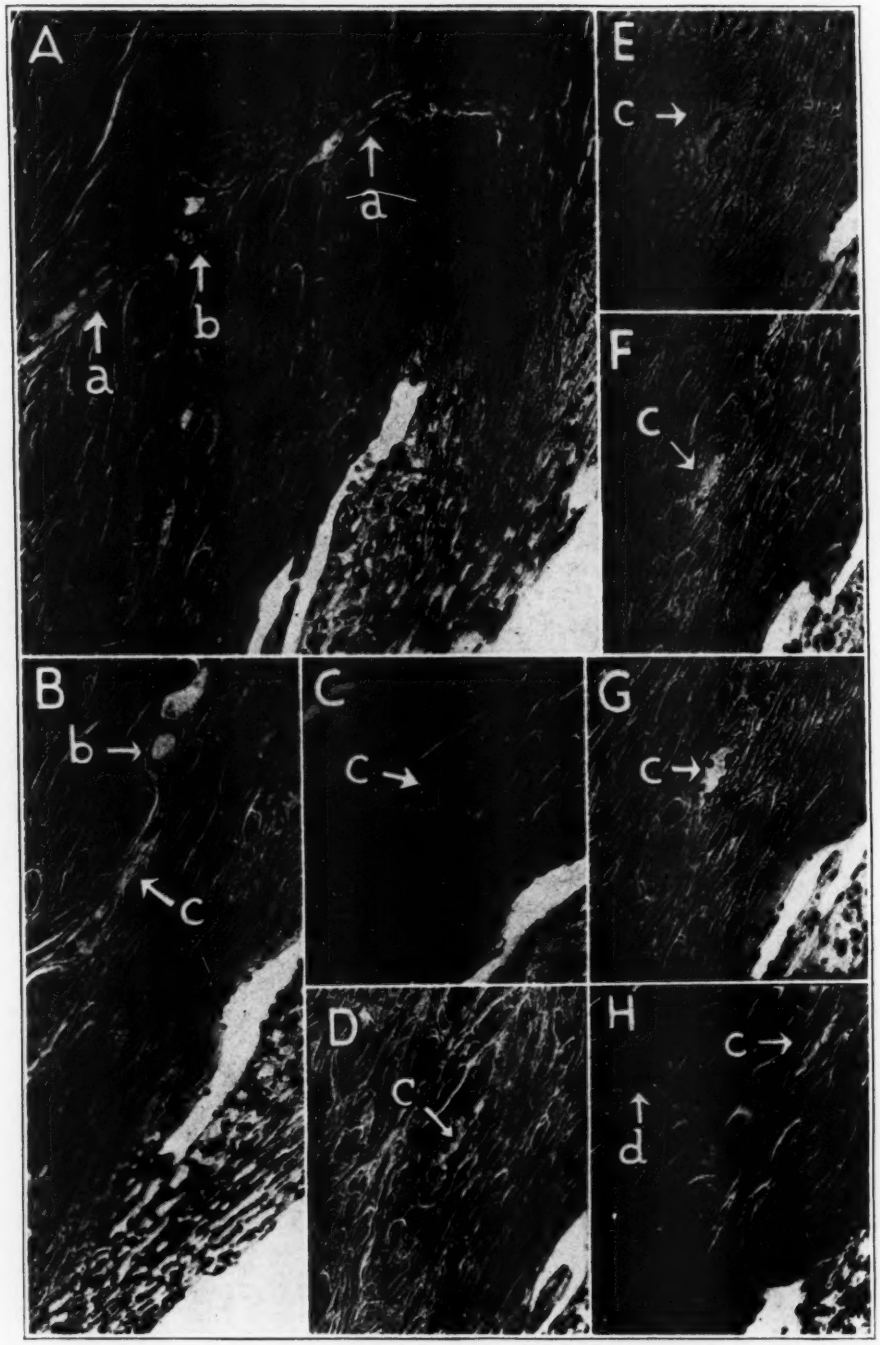

Fig. 4.—Photomicrographs selected from seventy-five successive serial sections in a case of absolute glaucoma in which degenerative changes had occurred in the vessels connected with Schlemm's canal. Compare with figure 6. An artery, *a-a*, which arises with its accompanying nerve from the ciliary body is seen in *A*. This gives off a smaller branch, *b*, which runs perpendicularly to the plane of the sections. In *B* this branch gives off a branch, *c*, which can be followed through *C* to *H*. In *E* and *F* the wall of *c* is necrotic and the lumen filled with a thrombus. In *H*, *c* bifurcates into *d* and *e* (*e* shown in fig. 5 *A*).

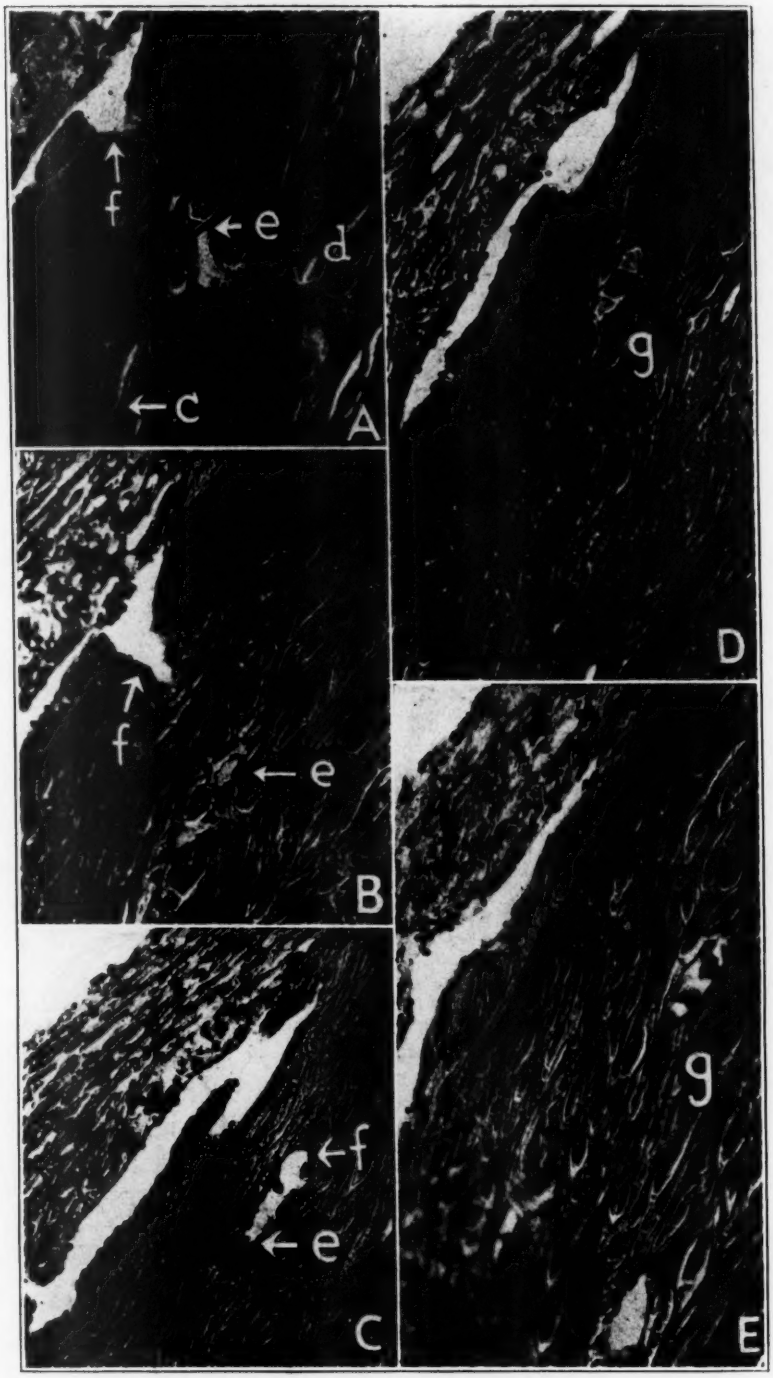

Fig. 5.—Branch *e* connects with an outgrowth of Schlemm's canal *f* in *C*, and a branch arising at this junction, *g*, shows a proliferation of its endothelium in *D* and *E*.

Up to the present only one case of early and uncomplicated simple chronic glaucoma has been available to me for histologic study. The patient was a white man of 73 who had been known to have glaucoma for eighteen months prior to his death. The highest recorded intra-ocular tension had been 26 mm. of mercury (Schiötz) in each eye, and under miotics the tension was reduced to 12 mm. The anterior chambers were of normal depth, the visual acuity was normal in each eye, but the optic disks showed marked glaucomatous excavations. The peripheral regions of the fields were normal, but one eye showed an enlargement of the blindspot of the Seidel type, while the other showed a marked Bjerrum scotoma. Three weeks after my last examination of this patient he died of pyelonephritis following prostatectomy.

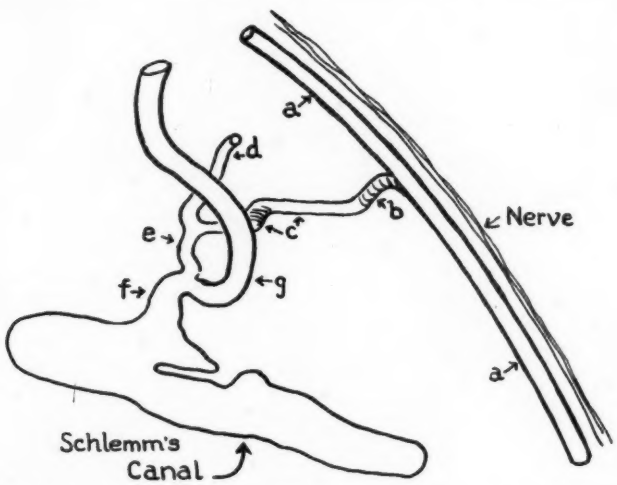

Fig. 6.—Schematic drawing showing the connections of the vessels shown in figures 4 and 5.

Examination of his eyes post mortem revealed the glaucomatous excavation of the disks with slight optic atrophy. There were no peripheral anterior synechiae. The ciliary body was normal. Schlemm's canal was patent, and the pectinate ligament was not sclerosed (fig. 5). The afferent arterioles of the canal showed marked hyaline thickening of their walls and reduction of their lumens. The degree of sclerosis of these vessels considerably exceeded that of the other intra-ocular arterioles. Aside from a moderate arcus senilis and some cystic degeneration of the anterior part of the retina, no other changes were found in the eyes.

It should be made abundantly clear that the observations in this single case, even though supported by similar observations in eyes showing the more advanced stages of the disease, are wholly inadequate to confirm the hypothesis as to the pathogenesis of chronic glaucoma which I have outlined. Furthermore, it cannot be assumed that there is

only one possible cause for the disease picture which is classified as chronic simple glaucoma. Nevertheless, it seems worth while to formulate this hypothesis and to report the confirmatory evidence, feeble though it is, because cases of uncomplicated early glaucoma so rarely present themselves for histologic study and the opportunity for confirmation or refutation of this hypothesis may fall into other hands than mine.

SUMMARY AND CONCLUSIONS

Schlemm's canal is not connected with the anterior chamber by any open channels or pores. It is connected with the intrascleral vascular plexus at the limbus by both afferent arterioles and efferent venules, the latter about four times as numerous as the former.

The effects of a continuous flow of plasma from the afferent arteriole into the canal and of the dilution of the plasma within the canal with water osmotically attracted from the anterior chamber are discussed. It is shown that, within limits, an increase in the flow of plasma into the canal leads to an increase in the rate of absorption of the aqueous and a lowering of intra-ocular tension. Similarly a reduction in the flow of plasma into the canal would be expected to lead to a decrease in the rate of absorption of the aqueous and a rise in the intra-ocular tension. It is suggested that in some cases of chronic simple glaucoma the primary lesion may be a sclerotic occlusion of the afferent arterioles. Such sclerotic changes were found regularly in cases of advanced glaucoma and also in one case of early uncomplicated chronic simple glaucoma.

ISAAC HAYS

PIONEER AMERICAN OPHTHALMOLOGIST

NATHAN FLAXMAN, M.D.

CHICAGO

Isaac Hays, the son of Samuel and Richea Gratz Hays, was born in Philadelphia on July 5, 1796. His father, a wealthy merchant engaged in the East India trade, brought up his family with all the culture and luxury which his means enabled him to command. Isaac acquired from the refinements of his early home life the courteous and affable manner which later distinguished him.

The early education of the youthful Hays was obtained as a pupil of the Rev. Dr. Samuel B. Wylie, an eminent divine and classical scholar of Philadelphia. At the age of 16 Isaac entered the University of Pennsylvania. Here he obtained the degree of Bachelor of Arts in 1816. The elder Hays desired to have his oldest son succeed him in the mercantile business. Although young Isaac yearned for a professional career, he obeyed his father and worked one year in the counting house. He then abandoned his business career since he had no taste for such a life. His father not only agreed to this but helped to start him in the medical profession.

The motives that inclined the 21 year old Isaac toward the study of medicine revolved about one man—Dr. Nathaniel Chapman. In 1817 Isaac became an office pupil of this outstanding practitioner. The man himself attracted the eager student. Chapman was the medical man of his day, a Virginian by birth, wealthy, cultured, with a magnetic personality and of the highest social standing. Everybody knew Chapman, and he knew everybody. The friendship formed between preceptor and pupil during those early days endured and grew firmer over a period of thirty-six years.

During the period that he was an office pupil of Chapman, Hays attended the medical school of the University of Pennsylvania. He obtained his doctor's degree from this institution in 1820. The faculty at that time consisted of Physick, Wistar, James, Dorsey, Coxe, Chapman, Hare and Gibson. Hays entered practice while he lived at the northeast corner of Eighth and Sansom streets. Exactly from whom or how he became interested primarily in the diseases of the eye was not apparent. At the time he entered the practice of medicine in Philadelphia in 1820, George Frick of Baltimore was the only doctor in the

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country limiting his practice to ophthalmology. The few general surgeons who were attempting to do ocular work were poorly equipped. Their general knowledge of anatomy of the eye was good, but their conception of pathology was poor. The methods of examination were limited to inspection with the unaided eye and to palpation. Hays was an ambitious young man, inspired by the success of Chapman and desirous of making a name for himself in an untouched and unlimited field. His deliberate nature, mathematical interest and precise work well fitted him for the practice of ophthalmology.

The first textbook on the diseases of the eye printed in the United States was an American edition of Saunders' text which was published in Philadelphia in 1821. Hays consumed the material in this book from cover to cover. Most of his time was devoted to laying a firm foundation for future work, and much leisure was spent in the study of ocular conditions. He was appointed to the staff of the first ophthalmic dispensary founded in Philadelphia, the third in the United States in the same year. This dispensary was in existence until 1825. When the fourth ophthalmic hospital, the Pennsylvania Infirmary for Diseases of the Eye and Ear, was opened on Feb. 8, 1822, he was appointed one of the surgeons; the others were Robert Griffith, George Wood and John Bell. In that institution he obtained the actual clinical experience necessary to balance the vast amount of theoretical knowledge gained in his first two years in practice. The first work of an American writer, George Frick, on diseases of the eye, was published in 1823. An extended review of this book, written by Hays, appeared in an 1824 issue of the *Philadelphia Journal of the Medical and Physical Sciences*, which was edited by Chapman.

The Academy of Natural Sciences elected Hays to membership in 1818, and shortly afterward he was made a member and chairman of the Publishing Committee, in which office he was earnest and indefatigable. His first medical paper, entitled "The Forces by which the Blood is Circulated," was delivered as a lecture to the Academy of Natural Sciences in 1823 and was published as the leading article in the *Philadelphia Journal of the Medical and Physical Sciences* in 1826. This paper summarized the successive steps in a complete knowledge of the subject up to that time.

During those early years in practice Hays gained and held the friendship of a number of practitioners of his own age. All of them, Samuel Jackson, Franklin Bache, Charles Meigs, Rene LaRoche, George McClellan, Hugh Hodge, John Mitchell, Francis Condie and George Woods, were hard-working, ambitious men, devoted students like Hays, good writers and fine teachers, with scholarly tastes, refined manners and cultivated intellects. These men, constantly acting and reacting on one another, stimulated each other's ambitions.

In the few years before other important decisions had to be made, Hays laid the foundation of high repute as an ophthalmic surgeon. He intended to publish a series of papers on various ocular diseases. This was very evident with the publication of the first article, entitled "Observations of the Inflammations of the Conjunctiva." [1] In the same year another long article appeared, "Inflammations of the Sclera," [2] and this was followed by an extensive treatise on "The Pathology and Treatment of Iritis." [3] The nature of these articles indicates that Hays began a systematic study of his own observations on the clinical material seen in the Pennsylvania Infirmary and the Philadelphia Dispensary.

Hays showed unusual ability in another line of work, medical journalism. While his extensive work on diseases of the eye was appearing in print, he had to make the most momentous decision that could possibly confront a rising young practitioner of 30 years. When he became associated with Chapman, Dewees and Godman in the editorial management of the *Philadelphia Journal of the Medical and Physical Sciences* in 1826, and, when late in 1827 he became the founder and editor of the *American Journal of the Medical Sciences,* Hays stood at the brink of two tremendously fertile and undeveloped fields. Well qualified in both, he firmly believed that by devoting the major portion of his time to medical journalism some improvement in ophthalmology would result. Therefore he resigned from the staff of the Pennsylvania Infirmary in 1827 and concentrated his efforts in placing the first national medical journal on a successful basis.

When Dewees' textbook on the "Practice of Medicine" appeared in 1830, the chapter on the diseases of the eye was contributed by Hays. This chapter covered a concise account of the anatomy, physiology and pathology of the eye and the treatment of its diseases in about 70 pages. Dewees considered Hays the best and most suitable person to write that chapter for the book, and in the preface he not only mentioned this but alluded to Hays' long and attentive study of the subject together with his experience.

In 1830 James Wills of Philadelphia left a bequest to found a hospital for the indigent blind and lame. A building was erected on Race Street between Eighteenth and Nineteenth streets. The hospital was opened on March 3, 1834, and continued its work on the same site to the present day. The first staff was composed of Isaac Parrish, Squier Littell, George Fox and Isaac Hays. After a few years the work of the Wills Hospital became entirely limited to the diseases of the eye. Hays was on service three months and off service three months, alternating with George Fox. For twenty years, from 1834 to 1854, Hays

1. Philadelphia J. M. & Phys. Sc. **13**:84, 1826.
2. Philadelphia J. M. & Phys. Sc. **13**:211, 1826.
3. Philadelphia J. M. & Phys. Sc. **14**:217, 1827.

served this institution. During this period he contributed reports on the cases seen in his service in this hospital to the *American Journal of the Medical Sciences.* The other attending ophthalmic surgeons did likewise, and this journal published clinical material on the diseases of the eye continuously, which, in addition to the selected abstracts on ophthalmology that Hays contributed to that section of the magazine, was the only ophthalmologic literature uninterruptedly available to all practitioners for over forty years.

Two outstanding contributions to ophthalmology were made by Hays in the case reports from the Wills Hospital. He first observed and reported noncongenital color blindness as a pathologic condition—the case of Mary Bishop, which ended in recovery.[4] Although he did not record the first case of astigmatism in America, and the fifth in the world, until 1854, he noted this condition as early as 1839. This same article of 1840 contains references to cases of astigmatism. In 1843 he edited an American edition of Lawrence's treatise on diseases of the eye. A letter from Lawrence acknowledging a copy of the volume contained the following paragraph:

> I return you my best thanks for the book, and at the same time beg to express to you how highly I am gratified at finding that you not only think my work worthy of republication in America, but that you should have condescended to act as editor, and to enrich my treatise with those valuable additions which might well have constituted a separate publication. I feel that I could not have received a higher compliment, and I shall always hold the circumstance in grateful remembrance.

The preparations of the successive editions of this work was to Hays a delightful task not only because it associated his name with that of perhaps the outstanding ophthalmic surgeon in England but because it gave him an opportunity, as he himself said in the preface of the second edition, of presenting the results of his own experience derived from more than a quarter of a century's devotion to the subject, during which period he had been attached to a public institution for the treatment of the diseases of the eye. In the third edition, in 1854, he first recorded the correction of astigmatism in two cases, the cylindric glasses having been ground by McAllister,[5] the Philadelphia optician, in 1853. Bumstead carried on this work in New York and Hasket Derby in Boston. On receiving a copy of the third edition of his book, Lawrence wrote a letter of thanks to Hays:

> I feel it a particularly fortunate circumstance that one so thoroughly conversant with the whole subject, and so used to literary composition, should have undertaken the troublesome task of making these additions to my treatise, pub-

4. Am. J. M. Sc. **26**:277 (Aug.) 1840.

5. Personal communication to the author from Dr. G. E. de Schweinitz.

lished in 1840, which are necessary to bring it up to the present state of knowledge and should have conferred upon it the new and interesting feature of so many beautifully executed pictorial illustrations.

An instrument, combining the advantages of a knife and a needle, for the discission of hard cataracts was devised by Hays. Although it became obsolete later, the instrument was at the time much employed by American oculists. This needle-knife was used for the operation of cataract "by solution or absorption," and Hubbell said that "not only did this instrument admirably serve the purpose for which it was designed by Dr. Hays, but it is still an excellent knife for discission of after-cataract." Hays also edited, with notes, the American editions of T. Wharton Jones' "Principles and Practice of Ophthalmic Surgery" in 1847.

These contributions certainly suggest that Hays kept his early interest in ophthalmic problems. When the tremendous task of editing the national quarterly, and later the associated monthly journal, allowed some leisure time, he delved into his first love. The failure of Hays to become the outstanding ophthalmologist in the United States was due to the fact that he never became a teacher of that branch of medicine. This was not on account of his natural timidity before an audience. It was purely an accidental occurrence. He was to deliver the introductory lectures on the diseases of the eye and ear at the Philadelphia Anatomical Rooms in College Avenue, later called Chant Street, in the winter of 1826, but the lectures were never given. The removal of Godman, the main projector of the enterprise and the lecturer on anatomy and operative surgery, to New York and Hays' acceptance of the editorial office with the *Philadelphia Journal of the Medical and Physical Sciences* probably prevented him from becoming the most thorough and accurate teacher of his day. In later years whatever he was called on to say was to the point and tersely expressed. He impressed his hearers with a sincere and balanced conviction of his opinions.

At the age of 38 years he married Sarah Minus of Savannah, Ga., and this happy union was eventually blessed with four children. The peace and content of his home contributed much to the diligent and steady performance of his increasing literary work. He was rarely out of Philadelphia, even in the summer, and he did not have a vacation for twenty years after he became editor of the *American Journal of the Medical Sciences.* Hays was proportionately built and had blue eyes and a well formed head with a finely chiseled profile and a beaming and benevolent countenance. In his manners he was emphatically a gentleman of the old school, bland, gentle and dignified, with a sweet and subdued voice and a warm sympathizing heart. He was an early riser and seldom required more than five or six hours of sleep. His jour-

nalistic work was usually done in the early morning or in the evening, thus leaving him the remainder of the day for his professional, literary and public duties.

The life and work of Hays—these were synonymous to him—aside from ophthalmology, consisted of editing the *American Journal of the Medical Sciences* for fifty-two years and affiliated journals for thirty-six years and of his tremendous effort in behalf of the medical profession, individually and collectively.

In 1854, at the age of 58 years, Hays gave up all clinical work to devote himself to literary work. This arrangement was only for a short time, as he soon reentered private practice. Black stated that in 1858 in ophthalmology Hays and Squier Littell did little or no other work in Philadelphia; all the general surgeons did ophthalmologic work. In the sunset of Hays' life the American Ophthalmological Society was founded, in 1864, and Herman Knapp began the *Archives of Ophthalmology*, in 1869. When the Philadelphia Ophthalmological Society was founded, in 1870, Hays was elected its first president, at the age of 74 years. His son, I. Minus, was associated with him in both the editorial and the ophthalmologic work from 1869 to 1879.

With the advance of age, Hays' strength gradually gave way. His mind was active and still carried keen interest in all his work. In February 1879, influenza was epidemic in Philadelphia, and he was one of the sufferers from this malady. He outlived this attack but never rallied from its effects or regained his strength, being confined to his residence at 1525 Locust Street. On April 12, 1879, Isaac Hays passed from this life, one of devotion and beauty, at the venerable age of 83.

No monuments were raised to this man who devoted his lifetime to the interests of his profession. In every medical library of the world his remarkable work stands—103 volumes of the first and finest national medical quarterly. This journal was more than just a magazine to him. It was a living thing, throbbing with the ideas and thoughts of ambitious, hard-working, struggling practitioners everywhere. His relation to the men who contributed to this journal and what the publication of papers in it meant to these persons was best expressed, indirectly, by J. Marion Sims. While suffering from severe diarrhea which had forced him to leave the South, Sims went to Philadelphia in July 1852 to see some friends, as he felt he was going to die.

> When I saw that I could not recover, I sent for my friend Dr. Isaac Hays to come and see me. He came very promptly. I explained to him the condition of my affairs, and said to him that I felt I was going to die. He said that he thought I had better take cod-liver oil, and not to give up.

When Sims sought and obtained a home at 89 Madison Avenue in New York in September 1853, with little money on hand, no friends,

no influence, no health and nothing to commend him to business, he said:

> Fortunately I had published my article on the treatment of Vesico-vaginal fistula a year before that, in the American Journal of the Medical Sciences, and the doctors had read it everywhere, and were very much surprised at the claims set up of rendering this troublesome and loathsome affection easily and successfully cured. They hardly believed it. Whenever I was introduced to any of the doctors, they all knew who I was by that article, and by my previous contributions to the medical literature.

All of Hays' work has endured. His effect on American ophthalmology, medical journalism and organization can be noted in the firm foundation on which the profession stands. His pioneer work in the ocular field has been considered. In his efforts in behalf of the medical profession Hays can be compared with the most prominent figure in American medicine, William Osler. Three men, Benjamin Rush, Isaac Hays and William Osler, stand foremost in this country in the three hundred years of its medical growth. The first two were native Philadelphians, and the third was an adopted son. Hays lacked the sparkling personality of Osler, but in his quiet unassuming way the work of fifty-two years, steady unrelenting labor, shaped American medicine to conform with his high ideals of the ethics and dignity of such noble work.

The selection of Hays by the editors and publishers of the *Philadelphia Journal of the Medical and Physical Sciences* in 1826 to the staff of the journal was neither an accident nor an experiment. Isaac Lea, a member of the house that published this journal, was well acquainted with Hays' work in editing the *Journal of the Academy of Natural Sciences.* The *Philadelphia Journal of the Medical and Physical Sciences* originated in 1820 with Chapman as editor, and in 1825 William P. Dewees and John Godman were added to the staff. In the following year Godman moved to New York. The other editors were unable to devote their time to the interests of the journal because of increasing professional activities. It became necessary to select another editor, and the friendship of Hays with Chapman and Dewees and his acquaintanceship with Godman, along with the knowledge of his work by the publisher, Lea, made Hays the unanimous choice for the position. An announcement to this effect, prepared by Godman, appeared in the *Philadelphia Journal of the Medical and Physical Sciences*:

> The Publishers, with entire approbation of the Editors, announce with pleasure that they have secured the valuable assistance of Isaac Hays, M.D., who henceforth will co-operate in conducting the Journal. Independent of this gentleman's practical experience and zealous devotion to the interest and honor of the profession, we consider him as a great acquisition, on account of his long experience and well earned reputation in the management of such a publication. The Journal of the Academy of Natural Sciences owes a very large portion of its success to

his exertions and attention to the business of its publication. In adding him to the editors we give an ample pledge of our determination to make every effort to merit a continuance of the liberal support it has uniformly received.

The name of Isaac Hays appeared as one of the editors on the cover of the second number of the thirteenth volume of the *Philadelphia Journal of the Medical and Physical Sciences* in February 1826. By the end of the year he was the sole editor, as all his associates had retired. The Philadelphia journal had been created by Chapman in a spirit of indignation at the remark of Sidney Smith made in an essay in the *Edinburgh Review:* "In the four quarters of the globe, who reads an American Book? Or goes to an American play? Or looks at an American Statue? What does the world yet owe to American Physicians or Surgeons?" To emphasize his feelings, Chapman, as long as he was editor, quoted that arousing remark on the title page.

Hays did nothing by excess or impulse. His was a well balanced character. The position of American medical journalism in 1827, thirty years after its birth, was very uncertain. Hays was aware of this. The *Philadelphia Journal of the Medical and Physical Sciences* was the thirteenth medical magazine to be published in the United States since 1797. Ten of the thirteen journals, all local in character, had expired prematurely by the time that Hays became an editor. The ninth journal, founded in 1812, the *New England Journal of Medicine and Surgery*, and the twelfth, founded in 1818, the *Medical Recorder*, as the *Philadelphia Monthly Journal of Medicine and Surgery* was called, were the only ones in print at that time besides the *Philadelphia Journal of the Medical and Physical Sciences*. Hays also was of the opinion that the position of a profession was reflected in its magazines. Therefore, he laid aside the local character of the *Philadelphia Journal of the Medical and Physical Sciences*, combined it with the *Medical Recorder*, discarded Sidney Smith's remark from the title page, gave the new quarterly the name of the *American Journal of the Medical Sciences*, selected a group of thirty-nine collaborators representing many of the best and prominent men in the various sections in the country and issued the first number of the first national medical journal in November 1827.

His platform was to advance the interests of medical science, to foster and develop native talent, to disseminate medical knowledge, to elevate the character and dignity of the profession and to supply a want deeply felt by the American practitioner. Anonymous contributions, as well as all personalities, he scrupulously excluded. Each number of the *American Journal of the Medical Sciences* was issued under three separate headings: the department for original communications,

a review and biographical department and a perioscopic department devoted to a condensed account of the recent progress in medical science in the entire world.

As an editor Hays possessed an enlightened, unbiased and deliberate judgment. His manner invited confidence. He had a firmness that was proof equally against the allurements of designing flattery and the exactions of overweening self-esteem as well as against the dictation of ill advised counselors and the injustice of hostile criticism. These influences never disturbed the serenity of his feelings. He did what he felt obliged to do, never rashly or impulsively, and not with a zeal that was blind to the right of judgment in others. He kept before him the fact that errors may be propagated fanatically by men who are honest in their convictions and aims. It is almost without example that this leading medical periodical which he edited for more than half a century was never known as the organ of a party and never contained an editorial line that betrayed personal animosity. Until 1841 Hays edited the *American Journal of the Medical Sciences* with the aid of collaborators. In that year he became the sole editor, his name appearing for the first time on the title page of a new series of the quarterly. It became affectionately known by practitioners everywhere as the "Hays Journal." Many of the early bound issues have that title instead of the real name of the journal imprinted on the binding. For the next twenty-eight years he carried on all of the editorial work alone.

The *Cholera Gazette* of 1832, a weekly, was edited by Hays. Cholera appeared in Europe in that year and spread to Canada and New York. The Philadelphia Board of Health appealed to the local medical society, and a committee was appointed to investigate the matter. The reports of this committee were published in the *Cholera Gazette*. This bulletin was the attempt of medicine to organize as effectively as possible to stop the plague by reaching the physicians and the public with all possible information on the subject.

The dignity and gravity of the quarterly journal prevented it from containing discussions on the problems which concerned the medical profession not directly connected with the art and science of medicine. Therefore, in 1843 Hays founded the *Medical News* as a monthly journal to keep physicians informed of all that transpired of general interest in the various medical societies and colleges of the country, and especially of everything that tended to advance or retard the course of medical education. Part of its columns were also devoted to exposing quackery in all phases and forms. In pursuit of this reformative work Hays only once found himself called on to answer for what he had edited. In 1846 a suit was brought against him by the author of a quack remedy for having declared in the *Medical News* that the pamphlet describing this medicine "displays quackery in its most unblushing

and undisguised form." The case was tried in the Supreme Court of the state, and the jury sustained the criticism of Hays by merely awarding nominal damages to his assailant. The *Bulletin of Medical Science*, edited by John Bell, commented on the case as follows: "We must not let the opportunity pass without recording the obligations under which the profession lies to Dr. Hays for his continual exposure of quackery and medical delusions." The *Medical News*, which afforded a ready field for the display of personal feelings, never exhibited them during the years that Hays conducted it, not even at times when there was sufficient ground in the misconduct of others to justify the severest type of criticism.

In 1874 he began the publication of the *Monthly Abstract of Medical Science*, the forerunner of subsequent journals of abstracts, but this was merged with the *Medical News* the year after the death of its founder. Gross made the prediction at the time of Hays' death that when the history of American medical literature is written an important place will be assigned to this, the greatest and most gifted medical journalist of the nineteenth century.

The *Medical News*, from its inception in 1843, continually devoted space to the striving of individuals for better medical education and to the work of medical societies to improve the teaching and licensing of practitioners. The part Hays took in the organization of the American Medical Association was directly in line with his editorial ideas on the subject. Early in 1846, when the Medical Society of New York sent out the call for the first convention, he was sent as a delegate by the Philadelphia Medical Society. He took an active part in the proceedings that opened the first meeting on May 5, 1846, in New York. As a member of the committee appointed to bring the subject of medical education before the convention, he presented the resolutions that proposed to create a national medical association and to adopt a uniform and elevated standard of requirements for the degree of Doctor of Medicine so that young men before being received as medical students would obtain suitable preliminary education and that the medical profession in the United States would be governed by the same code of medical ethics. The following account of the origin of these resolutions was noted in an original manuscript of Hays:

> The idea of taking advantage of that occasion to form a National Medical Association is due, I have reason to believe to Dr. A. Stillé of Philadelphia. He consulted one of his colleagues (Dr. Hays) in regard to a series of resolutions which he had prepared to offer for the consideration of the convention; but as no opportunity for his offering them had presented itself, so great was the disorder on the first day of the meeting, a colleague (Dr. Hays), who had been appointed one of the committee to prepare the business for the next day, asked to be allowed to bring them before that committee and try to obtain the recommendation of the

passage of these resolutions. With some difficulty this was obtained, and the resolutions adopted by the convention were essentially the same as those prepared by Prof. Stillé.

These subjects were referred to several committees, and Hays was appointed a member of the one to prepare a code of medical ethics. In the meanwhile the medical journals published the proceedings of the convention in New York. This helped bring the subject to the attention of a large number of the members of the profession. Hays, as chairman of the Committee of Arrangements, welcomed the delegates to the meeting in Philadelphia on May 5, 1847, at the organization of the association. His report on the subject of medical ethics which was the work of the entire committee was very full and explicit. He stated that justice required that some explanatory remarks should accompany the code which was unanimously adopted by the convention:

> The members of the Convention would not fail to recognize in parts of it, expressions with which they were familiar. On examining a great number of codes of ethics adopted by different societies in the United States, it was found that they were all based on that by Dr. Percival, and that the phrases of this writer were preserved, to a considerable extent in all of them. Believing that language so often examined and adopted must possess the greatest of merits for such a document as the present, clearness and precision, and having no ambition for the honors of authorship, the Committee which prepared this code have followed a similar course, and have carefully preserved the words of Percival whenever they convey the precepts it is wished to inoculate. A few of the sections are in the words of the late Dr. Rush, and one or two sentences are from other writers. But in all cases, whenever it was thought that the language could be made more explicit by changing a word, or even part of a sentence, this has been unhesitatingly done; and thus there are but few sections which have not undergone some modification; while, for the language of many and for the arrangement of the whole, the Committee must be held exclusively responsible.

Gross expressed the opinion that the code may not have been faultless, but it was as free from errors as it is possible for any code of morals to be. Hays was elected treasurer of the convention out of which grew the American Medical Association and was annually reelected from 1848 to 1852, when he declined being again a candidate for the office.

As chairman of the Special Committee on Representation at the sixth meeting in New York in 1853, Hays objected to the inequality in the ratio of representation and presented a remedy in the form of member to delegate ratio. He proposed that the Association be composed solely of members elected directly by it or through its Council and that this Council should be a permanent body, renewable in part each year, the duties of this Council to be the general superintending of the affairs and publications of the Association. These suggestions were not adopted. Later, by amending the law in regard to representation, the convention imperfectly accomplished the object aimed at by

Hays in the early sessions. Some of the early difficulties of the Association can be traced to the tardy acceptance of his ideas on representation. Members were elected on the basis of personal popularity and not on their professional merit or distinction. It seems probable that part of the disinterest shown in the Association for many years by the more cultivated members of the profession and the medical colleges was also due to this system of election. Many years elapsed before all of Hays' suggestions on the maintaining of separate sections for the discussion of general and specialized subjects and the creation of a Judicial Council to handle all questions of an ethical or judicial problem were carried out in full content.

Hays became a fellow of the College of Physicians of Philadelphia in 1835 and performed active work in its behalf. He was chairman of the building committee, took an especially active interest in the library and edited the transactions of the college for a number of years. In 1848 he was a delegate from the college to the convention for organizing the Medical Society for the State of Pennsylvania and the chairman of the committee which drafted the constitution for that society. The best way to summarize his organization work in behalf of medicine would be to restate that he was among the founders of more than a dozen associations, the oldest member of a dozen more and the chief officer of another dozen societies.

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MARGINAL DYSTROPHY OF THE CORNEA ASSOCIATED WITH PROLAPSE OF THE IRIS

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PHILADELPHIA

The ocular condition present in the patient whose case is to be reported has been described under various names, furrow keratitis, marginal degeneration of the cornea, ectatic marginal dystrophy of the cornea and other variants. Marginal dystrophy of the cornea seems most appropriate, as this feature is common in all cases and the term is in keeping with modern nomenclature.

The disease is rare. About 75 per cent of the reported cases were in males between the ages of 10 and 70 years. The condition is usually bilateral but is not always in the same stage of development in the two eyes. There is often a history of irritation of the eye and sometimes of persistent slight conjunctivitis. Rarely, rupture of the globe brings the first subjective symptom, or this complication may occur without subjective symptoms, as in the present case.

The furrow usually develops in the upper part of the cornea and may extend around the full circumference. It is preceded by opacity and peripheral vascularity. The opacity is in the form of a gray line. The furrow develops between this line and the margin of the cornea. The epithelium is intact. With the deepening of the groove the central edge becomes abrupt, and the floor either presents one or several localized ectatic areas or becomes ectatic in its entire extent as the result of the yielding of the thinned tissue to the intra-ocular tension. Seldom does ectasia occur in the inferior half of the cornea. The occurrence of ectasia is often delayed for many years. At several points the conjunctiva may be drawn over the area of ectasia in the form of pseudopterygia. In the area of the groove sensibility is absent. The intra-ocular tension is, as a rule, normal. Rupture of the floor of the groove, with resulting prolapse of the iris, usually follows slight trauma, though it may occur spontaneously, as is probably the case when the prolapse is bilateral. In Uhthoff's case, however, though the rupture was bilateral and occurred many years apart in the two eyes, it was attributed to a slight injury in both instances. In time the bulbar conjunctiva covers the protruding iris.

Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, April 16, 1936.

The alteration in the curve of the cornea diminishes visual acuity, and this is due in nearly all cases to the development of a high myopic astigmatism against the rule—in Ischreyt's case, 22 D. In several cases, including one recorded by Parsons, and in the case I reported in 1911, crystals have been present either in the uninvolved portion of the cornea or within the furrow. Handmann frequently observed degenerative changes in the macula.

Rupture of the cornea seems to be a rare complication. In Uhthoff's case it occurred in both eyes. In Schutz' patient, a boy aged 18 years, both eyes showed peripheral ectasia and in one eye rupture had occurred. Manes and Moulié recorded a case in a man, aged 37, in both of whose eyes there was prolapse of the iris. The herniated iris was covered with conjunctiva drawn onto the cornea.

Axenfeld and Gifford each performed a successful operation for the removal of cataract in an eye presenting marginal dystrophy. Both operators covered the incision with a conjunctival flap. Gifford first applied tri-chloracetic acid.

The cause of the condition is unknown. According to Doggart, bacteriologic studies have thrown no light on the cause. Trachoma, iridocyclitis and glaucoma have been associated with the condition, and in one case there was a history of malnutrition. In both of Cattaneo's cases hypercholesteremia was shown, and Bohm and Fassel noted hepatic hypofunction in both their cases. Lugli suggested endocrine dysfunction.

In a case of Parson's, reported by Doggart, a study was made with the slit lamp. In the left eye endothelium was not visible in the middle portion of the ectatic zone, but its amorphous remains appeared in the lateral portion. Immediately below the two ends of the white line, the corneal stroma, for a distance of 1 mm., showed abundant glistening crystals and ill defined grayish opacities. In the lower portion of the cornea of this eye and the upper portion of the cornea of the right eye, which appeared normal on examination with the naked eye, conjunctival vessels encroached at various points for a distance of 1 mm., and a number of scattered grayish dots of opacity appeared in the substantia propria. No thinning of the cornea was yet demonstrable. These phenomena presumably denoted the beginning of the process the later stages of which were evident in the upper portion of the periphery of the cornea of the left eye.

The anatomic change which has constantly been noted is complete degeneration of the substantia propria and of Bowman's membrane. This is preceded by separation of their fibers and splitting of the lamellae of the cornea into loose fibrillar, richly nucleated tissue. The ends

of the fibers which reach the affected area are clubbed and wedge-shaped and are transparent. Fat globules are observed in the corneal lamellae in the vicinity of the furrow. These changes represent local degeneration of the corneal tissue and an effort to compensate for the loss of the corneal tissue by the formation of fibrillar tissue rich in nuclei and blood vessels. As this attempt is only partially successful, thinning of the diseased area of the cornea results, which manifests itself clinically in the formation of a furrow.

Instances of prolapse of the iris have been reported by Uhthoff (two cases), Seefelder (two cases), Schutz (one case) and Drüner and Wiedersheim (each one case). Fuchs, Seefelder, Rupprecht, Kuryama and Coats have made histologic examination.

According to Gifford, who has reported several instances of marginal corneal dystrophy, the only others reported in the United States prior to 1924 were the cases of Knapp, Schutz, Gonzalez and Zentmayer.

Doggart has completed the list of references to 1930. To these must be added the reports of Manes and Moulié,[1] Bohm and Fassell,[2] Lugli,[3] Höhr Castán [4] and Opin and Reboul.[5]

REPORT OF A CASE

On Oct. 21, 1935, Mrs. C. F., aged 52, was referred to me by Dr. M. E. Smukler. The patient was born in Russia and came to the United States fourteen years ago. There was a history of inflammation of the eyes during childhood. In 1930 she was treated for hyperthyroidism. The same year Dr. Smukler performed multiple ignipunctures for the relief of entropion and trichiasis of the lower lid of the left eye. Shortly before the patient was seen by Dr. Smukler, she had consulted a druggist because of a foreign body in the right eye. In removing the body he noted the prolapse of the iris.

Examination of the patient revealed trichiasis of the lower lid of the left eye and slight ptosis of the upper lid of the right. On both sides the sulcus of the superior lid was deep, so that a large part of the upper surface of the eyeball was covered by the lid. In both eyes there was marginal dystrophy of the entire circumference of the cornea. The resulting furrow was of uneven depth. In the right eye there was an area of prolapse of the iris about 3 mm. in diameter

1. Manes, A. J., and Moulié, H.: Arch. de oftal. de Buenos Aires **5**:5 (Jan.) 1930.

2. Bohm and Fassell: Sborn. oftal **5**:174, 1930.

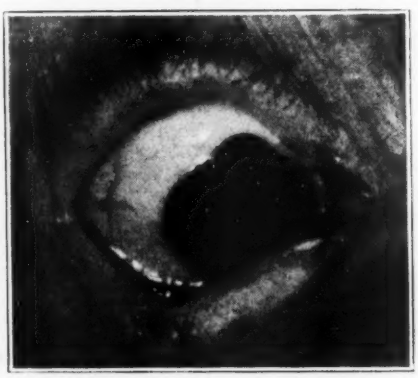
3. Lugli, L.: Arch. di ottal. **38**:449 (July-Aug.) 1931.

4. Höhr Castán, J. M.: Degeneración marginal de la córnea, Arch. de oftal. hispano-am. **35**:74 (Feb.) 1935.

5. Opin and Reboul: Dystrophie marginale des cornées chez les jeunes sujets, Arch. d'opht. **52**:771 (Nov.) 1935.

through the floor of the furrow (figure). The protruding tissue of the iris was covered with epithelium. The sensibility of the cornea was subnormal. The epithelium was intact, as evidenced by the absence of staining with fluorescein. The tension in the right eye was 22 mm. and in the left 20 mm. A 1 per cent solution of physostigmine salicylate was instilled into the eye, with little or no effect on the size of the prolapsed area.

Biomicroscopic Examination.—As the corneal image of the light was swept across the limbus, it became constricted in the grooved portion. The constriction was particularly marked at the upper margin where a deep groove reached well into the substantia propria. The epithelium was intact. The vascular loops of the limbus extended farther into the cornea than normally. It was impossible to determine whether the endothelium was intact in the involved portion of the cornea, but it was normal in the surrounding area. No folds were visible in Descemet's membrane. There were disseminated areas of thinning of the uveal pigment in the zone of the sphincter. The lens was normal. The biomicroscopic observations were corroborated and elaborated by Dr. Alfred Cowan.



Photograph showing marginal atrophy of the cornea, with prolapse of the iris.

The error of refraction of the right eye was corrected by $-3.00 \bigcirc +5.50$ cyl., axis 180°, with a vision of 6/9 part, and the error of the left, by $-0.75 \bigcirc +4.50$ cyl., axis 90°, with a vision of 6/6 part.

Stated in terms of crossed cylinder equivalents, the correction for the right eye was —3.00 cyl., axis 90° $\bigcirc$ +2.50 cyl., axis 180°, and for the left eye, —0.75 cyl., axis 180° $\bigcirc$ +3.75 cyl., axis 90°. It is thus clear that in the right eye the myopic meridian was the higher and the astigmatism was against the rule, which is the usual finding in this condition, while in the left eye the hyperopic meridian was the higher and astigmatism was with the rule.

In 1911 I presented before a meeting of this section a case of this condition in a woman, aged 48. The condition was bilateral, with a well marked groove encircling the margin of the cornea except for an arc of about 15 degrees in the lower part of the inner portion. It lay peripheral to an area which was not uniformly dense and on which the furrow encroached in places. In several places small pseudopterygia and at other points yellowish dots, presumably hyaline granules, were present.

PRIMARY TUMOR OF THE OPTIC NERVE

REPORT OF THREE CASES

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Primary tumor of the optic nerve is not common, although in the literature of the past sixty-four years about 300 cases of tumor of the optic nerve have been reported. Four have been observed at the Massachusetts' Eye and Ear Infirmary in Boston in thirty-six years, and one, at the Wills Hospital in Philadelphia in twelve years. Hudson [1] found that this type of tumor was more common in females than in males and that 75 per cent of the patients were in the first decade of life, which led some authors to suggest that the neoplasm is congenital.

The tumor may spread from the intra-orbital to the intracranial portion of the optic nerve through the optic foramen or from the intracranial to the intra-orbital portion of the nerve. In possibly the majority of the cases reported the neoplasm has occurred in the intra-orbital portion of the nerve. An intra-orbital tumor of the optic nerve can be diagnosed with a fair amount of certainty. Usually the first symptom is a gradual monocular loss of vision in a young person (the patient being under 21, as a rule), followed by a painless proptosis of the globe on the affected side. The proptosis is in the orbital axis, and there is resistance when attempts are made to palpate the globe backward. Ocular movements are but slightly impaired. The fundus shows changes which are confined chiefly to the disk, such as papilledema or atrophy of the optic nerve, Of 118 cases reported by Hudson,[1] in only 3 was the fundus normal. In one half of the cases the fundus showed atrophy of the optic nerve, and in the other half it showed papilledema. Roentgenograms of the optic foramen are often of value as a diagnostic aid in these cases. The foramen may be enlarged considerably, as in case 1 of this series. Whitnall [2] gave the average diameter of the canal as 5.5 mm. for adults. The average for children is about from 4.5 to 5 mm.

From the Department of Ophthalmology, University Hospital, University of Michigan Medical School.

1. Hudson, A. C.: Primary Tumors of the Optic Nerve, Roy. London Ophth. Hosp. Rep. **18**:317 (July) 1912.

2. Whitnall, S. E.: Anatomy of the Human Orbit and Accessory Organs of Vision, New York, Oxford University Press, 1932.

The intracranial primary tumor of the optic nerve or chiasm cannot be diagnosed so easily. Martin and Cushing [3] were able to diagnose such a tumor preoperatively in only 1 of 7 cases. There is usually no exophthalmos unless the intra-orbital portion of the nerve is involved. Loss of vision is slowly progressive in one or in both eyes. Observation of the visual fields is important and may show bitemporal or homonymous hemianopia or total blindness in one and hemianopia in the other eye. Roentgenograms may show deformation of the sella turcica and enlargement of the optic foramen. The fundus may show papilledema or atrophy in one or in both eyes.

The prognosis in these cases depends on the location of the tumor. If the intra-orbital portion of the nerve is involved and the tumor can be removed completely, prognosis for life is good. If the intracranial portion of the nerve is affected, the prognosis for life is poor, even though all the tumor can be removed. Martin and Cushing [3] operated on 7 patients and only 1 survived. The possibility of recurrence is remote if all the tumor has been removed.[4]

The best method of treatment for such a tumor is surgical, although roentgenotherapy has been used with varying results in treating intracranial neoplasms when it was impossible to remove all the tumor. For the intracranial and for some intra-orbital tumors the cranial or frontal approach is best. This consists of removing an osteoplastic flap from the frontal region and elevating the frontal lobe of the brain. An intra-orbital tumor can be removed by the Krönlein method of resecting the lateral wall of the orbit.

REPORT OF CASES

CASE 1.—C. M., a girl aged 10 years, was admitted to the University Hospital on Nov. 13, 1934. For five weeks poor vision and a slight bulging forward of the left eye had been noted. The proptosis had gradually increased.

The results of physical examination were essentially negative.

Ophthalmologic examination gave the following results: Vision was 6/9 in the right and 6/30 in the left eye, and the tension to the fingers was normal in each eye. There was definite gross exophthalmos of the left eye; the protrusion as measured with the exophthalmometer amounted to 13 for the right and 18½ for the left eye. The eyes were straight and the extra-ocular movements normal. The conjunctiva were normal in both eyes; the pupils were equal in size and round, the left reacting more sluggishly to light than the right. The globe was displaced forward but in no other direction, and it could not be pushed backward. There was no tenderness; no abnormal orbital mass was felt, and no bruit was heard. Examination of the fundus of the right eye gave negative results; that of the left eye showed papilledema of from 3 to 4 diopters. No hemorrhages were seen. The veins were normal in the right and engorged and moderately tortuous

3. Martin, P., and Cushing, H.: Primary Giomas of the Chiasm and Optic Nerves in Their Intracranial Portion, Arch. Ophth. **52**:209 (May) 1923.

4. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of the Optic Nerves in Their Intracranial Portion, Arch. Opth. **52**:209 (May) 1923.

in the left eye. The right visual field was normal; the left showed slight concentric contraction of form and color and enlargement of the blindspot.

Otologic and neurologic examinations gave negative results.

Roentgen examination showed minimal increase in the size of the left orbit and moderate enlargement of the left optic foramen.

Operation.—A diagnosis of tumor of the left optic nerve was made, and on November 24 the patient was operated on in the department of neurosurgery. A flap of the scalp was turned down in the left frontal region, an osteoplastic flap removed, the dura incised and the left frontal lobe elevated, exposing the intracranial portion of the left optic nerve, which appeared normal. The roof of the orbit was then removed, and the fatty tissue over the orbit bulged upward. When this was separated a large tumor, 15 mm. in diameter, almost globular, of bluish, almost translucent color was exposed. It appeared cystic, but aspiration revealed no fluid. There was only about 3 mm. of optic nerve between the eyeball and the mass, and this was enlarged. The optic nerve was divided close to the eyeball and the nerve found to be enlarged to the optic foramen. Therefore the foramen was opened and the nerve divided intracranially. The dura was then closed, the osteoplastic flap returned to its position and the incision closed.

Postoperative Course.—Recovery was uneventful. There were a definite pulsation and complete ophthalmoplegia with ptosis of the left eye. The disk was white, and there were hemorrhages about it. The veins were enlarged, and there were breaks in the blood column of the arteries. There were extensive pigmentary changes in the periphery of the retina. A few days later the fundus showed a more pink color. Two months later, on Feb. 4, 1935, the patient was in excellent health and had had no symptoms relative to the operation. The right eye was normal. The left eye showed a complete ptosis, with some function of the sixth nerve but none of the third and fourth nerves. Readings with the exophthalmometer were 16 for the left and 14 for the right eye. The fundus of the left eye showed a proliferation of vessels at the disk; the retinal vessels were replaced by white streaks, and the macula appeared grayish pink. In the periphery there were large areas of dense pigmentation and atrophy of the retina. When the patient was seen shortly before this report was made she was in good health.

Pathologic Diagnosis.—Examination of the tumor in the department of neuropathology revealed that the growth had invaded the perineural sheaths as well as the nerve itself. It consisted of unipolar and bipolar spongioblasts, forming a dense irregular and interwoven network. A diagnosis of spongioblastoma polare was made.

CASE 2.—R. A., a boy aged 6 years, was admitted to the University Hospital on Feb. 18, 1933. Two and one-half years before the child was admitted the mother noticed that the left eye was beginning to bulge outward. This bulging was more marked after the patient had had measles two years before he was seen. There was no complaint of blindness, but it was discovered that vision was defective in the left eye. The child had always been nervous. Recently he had complained of pain in the region of the forehead, especially in the morning. There had been no nausea or vomiting. The past history and the family history were irrelevant.

Physical examination and examinations of the blood and urine gave negative results.

Ophthalmologic examination showed vision to be 6/9 in the right and nil in the left eye. Tension to the fingers was normal in each eye. The pupillary reflex to light was lost in the left eye. There was definite exophthalmos, the protrusion as measured with the exophthalmometer amounting to 13 in the right and 18 in

the left eye. The left eye was proptosed in the orbital axis, and there was resistance to backward pressure. No mass could be palpated about the globe. In the fundus of the right eye the veins were slightly full but the appearance was otherwise normal. Examination of the fundus of the left eye showed that the media were clear, the disk was pale and had an infiltrated appearance and there was papilledema of 1 diopter. The veins were full and the arteries normal.

Roentgen examination showed definite alteration in the density of the posterior wall of the left orbit. There was also marked enlargement of the left optic foramen.

Neurologic examination gave no evidence of an intracranial lesion.

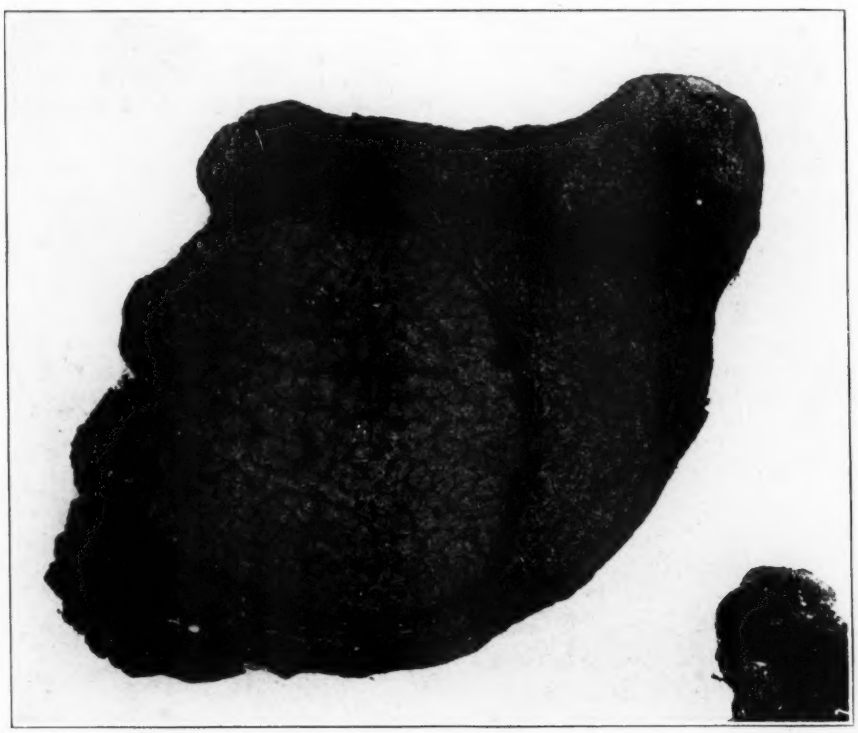

Fig. 1 (case 1).—Low power magnification of a cross-section of a spongioblastoma unipolare of the optic nerve, showing the tumor involving the nerve and perineural sheaths (azan stain, microplanar 35 mm.).

Operation.—A diagnosis of glioma of the left optic nerve was made, and on April 25 the patient was operated on in the department of neurosurgery. A flap of the scalp was turned down in the region of the frontal and parietal bones on the left, an osteoplastic flap removed and the dura incised. The left frontal lobe was elevated, a fibrous tumor of the left optic nerve being exposed. The tumor had passed through and posterior to the optic foramen; it was 1.5 cm. in diameter and of a bulbous appearance, being smaller toward the chiasm. It was believed to have extended into the left optic tract. The roof of the orbit was removed, and the tumor was found to extend to about 0.5 cm. from the globe. The nerve was divided on a level with the globe; the chiasm was divided close to the right

optic nerve, and the tract was divided back of the chiasm to get all the tumor tissue possible. The dura was closed, and the osteoplastic flap and flap of scalp were replaced.

Postoperative Course.—The convalescence was somewhat prolonged owing to low grade meningitis. This necessitated frequent lumbar punctures for several weeks. Five months later the patient's physician reported that vision in the right eye was nearly normal. Temporal hemianopia was present on the right because the left optic tract had been cut. The left eye was slightly lower in the orbit than the right. In the last report, on Sept. 11, 1934, the patient's physician stated that vision was normal in the right eye and that the patient was in good health.

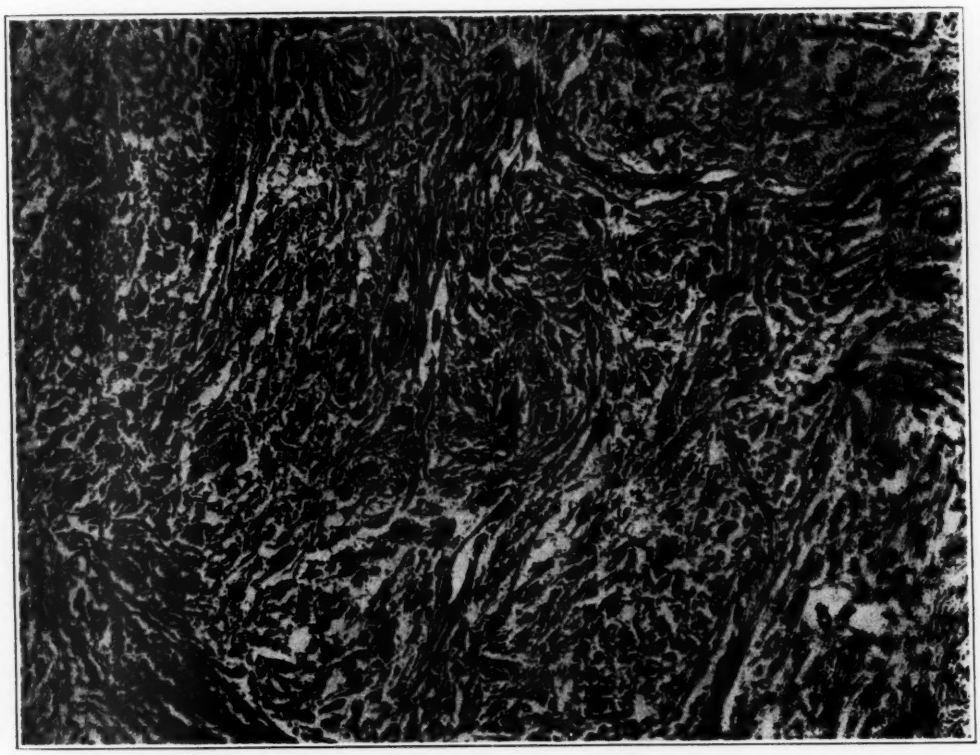

Fig. 2 (case 2).—High power magnification of a section of a spongioblastoma unipolare of the optic nerve. The unipolar spongioblast cells are cut in longitudinal and cross-section. The interwoven character of the tumor is clearly seen (phosphotungstic acid-hematoxylin stain, 0.16 mm.).

Pathologic Diagnosis.—The tumor was examined in the department of neuropathology. It was described as invading the optic nerve diffusely, bringing about its almost entire disintegration. The elements were unipolar spongioblasts in an early stage of development. There were still preserved bundles of nerve sheaths which were surrounded by a spongioblastic network frequently rich in fibers. The tumor tissue contained no glial tissue. There was no tendency toward necrosis. Lipoids were practically absent. A diagnosis of spongioblastoma unipolare was made.

Case 3.—W. J., a boy aged 15, was admitted to the University Hospital on April 20, 1933. He complained of poor vision in the left eye, which had apparently not become worse since its onset six years before. There had been nausea and vomiting during the past two or three years, the vomiting being questionably of a projectile type. The patient was seen six years before by his home physician and given corrective glasses because of poor vision. The past history and the family history were irrelevant except for the fact that the patient had malaria at the age of 11 years.

Physical examination showed the patient to be well developed and not acutely ill.

Ophthalmologic examination showed the extra-ocular movements to be normal except for a constant, although irregular, vertical, oscillatory type of nystagmus. There was no deviation under cover, and the near point of convergence was normal. The conjunctiva, cornea, anterior chamber and iris were normal. The pupils were round and regular and reacted normally to light but sluggishly in accommodation. The fundi showed the media to be clear; the right disk showed diffuse atrophy with but little infiltration; the arteries and veins were normal; the maculae were

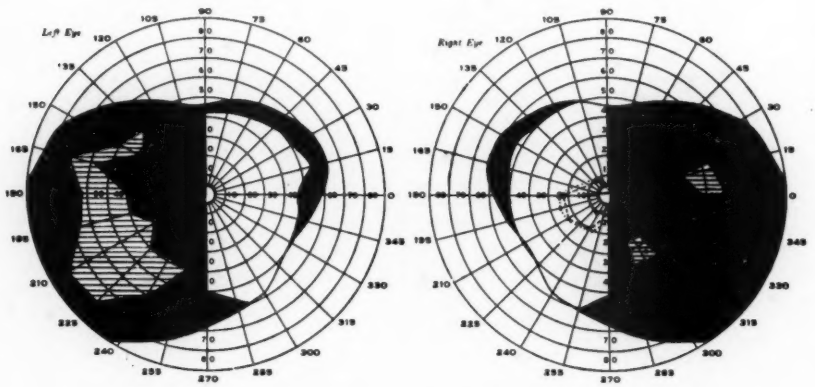

Fig. 3 (case 3).—Visual fields, showing bitemporal hemianopia with islands of vision in the temporal fields. The islands of vision are shown by the shaded areas. Vision was 6/6— in the right eye, and fingers were counted at 1 foot with the left. The test objects used were as follows: 5 mm. at 0.25 meter, 5 mm. at 1 meter and 10 mm. at 0.25 meter. In the right visual field the solid line indicates vision for green; the line of dashes, vision for red, and the dotted line, vision for blue. The colors were not seen with the left eye.

distinct, and no peripheral lesions were seen. The left disk was more atrophic than the right. Vision was 6/6— in the right eye; there was ability to count fingers at 1 foot (30 cm.) in the nasal field in the left eye. The visual fields showed bitemporal hemianopia with islands of vision in the temporal fields.

Roentgen examination was reported to show an increase in the prominence of the digital markings of the cranial vault and erosion of the sella turcica.

Operation.—A tentative diagnosis of pituitary tumor was made, and on April 21 an intracranial operation was performed in the department of neurosurgery. A flap of scalp and an osteoplastic flap were removed on the right side, the frontal and anterior part of the parietal region being exposed. When the dura was incised and the right frontal lobe elevated, a thin capsulated tumor about 35 mm. in diameter covering the sella turcica was exposed. The right optic nerve was

pressed slightly laterally, and the tumor arose from the left optic nerve which was entirely replaced with neoplastic tissue nearly up to from 1.5 to 2 mm. from the optic foramen. The nerve was divided at the optic foramen and a large portion of the tumor removed. The entire optic chiasm had been replaced by neoplastic tissue, but the right optic nerve showed no evidence of tumor, and hence was separated from the chiasm. The tumor extended back as far as the pons; hence no attempt was made to remove all the tumor. The dura was closed, and the osteoplastic flap and the flap of scalp were replaced.

Postoperative Course.—The patient did not recover consciousness and died twenty-four hours later. The temperature was 105 F. before death.

Pathologic Diagnosis.—The tumor was examined in the department of neuropathology and found to consist of elongated unipolar spongioblasts. The growth of connective tissue was moderate. A diagnosis of spongioblastoma unipolare was made.

COMMENT

The most common type of primary tumor of the optic nerve is the so-called glioma of subdural origin. Hudson [1] in 1912 stated that of 154 cases reported in the literature 118 were instances of glioma although only 28 were reported as such. In the past this type of tumor has been described as myxoma, fibroma, fibromyxoma, fibrosarcoma and myxosarcoma, probably because it was thought to be of mesodermal origin. Grinker [5] and DeLong [6] expressed the belief that all tumors of the optic nerve (gliomas) are of the spongioblastic unipolar or bipolar type, as in the 3 cases reported here.

Almost the only other type of tumor of the optic nerve is the endothelioma (if this can be considered, strictly speaking, a tumor of the optic nerve), which is of mesoblastic origin. The endothelioma originates in the nerve sheath surrounding the nerve and causes gradual atrophy. The differential diagnosis of glioma or spongioblastoma and endothelioma is not easy. In cases of spongioblastoma there is usually first blindness and then exophthalmos, while in cases of endothelioma the reverse is true. Restriction of ocular movements is more common in cases of endothelioma. The age of incidence in cases of endothelioma is greater, about 50 per cent of tumors of that kind occurring in patients over 30. In cases of endothelioma there may be circulatory obstruction shown in the lids and conjunctiva.

Neurofibroma of the orbit has been reported, but it is rare (4 cases are reported in the literature). The findings depend on the location of the tumor in the orbit and may easily be confused with signs of a tumor of the optic nerve. The neurofibroma is thought to arise from one of the various cranial nerves of the orbit. It commonly resembles a tumor

5. Grinker, R. R.: Tumors of the Optic Nerve, Arch. Ophth. **4**:497 (Oct.) 1930.

6. DeLong, P.: Primary Tumors of Optic Nerve: Report of a Case, Am. J. Ophth. **17**:797 (Sept.) 1934.

of the optic nerve when located deep in the orbit; when it is more shallow the globe is likely to be proptosed in an upward or downward direction, in contradistinction to straight forward.

SUMMARY

Primary tumor of the optic nerve is rare. It occurs more commonly in females, and 75 per cent of the patients with such a tumor are seen in the first decade of life. The tumor may be in the intra-orbital or in the intracranial portion of the nerve or in both. If it is in the intra-orbital portion it is more easily diagnosed. The symptoms are a gradual monocular loss of vision, usually a painless proptosis of the globe in the orbital axis and papilledema or atrophy of the optic nerve. A roentgenogram often shows an enlarged optic foramen. If the tumor is in the intracranial portion of the optic nerve there is a gradual loss of vision in one eye or in both eyes, without proptosis. The visual fields show bitemporal or homonymous hemianopia or total blindness in one eye and hemianopia in the other. A roentgenogram may show deformation of the sella turcica. The prognosis in cases of intra-orbital tumor is fairly good if the tumor can be removed, but in cases of intracranial tumor it is poor because of operative risk and extension of the tumor to vital parts of the brain. In the 3 cases reported here the tumor was of the spongioblastoma unipolare type.[7]

7. Permission was granted by Dr. Max Minor Peet, of the department of neurosurgery, for the use of the operative report in each case and by Dr. Konstantin Lowenberg, of the department of neuropathology, for the use of the pathologic description in each case.

Clinical Notes

AN IMPROVED PERIMETER-CAMPIMETER

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A combination perimeter and campimeter is described which is designed for every-day clinical work. It takes a middle course between the very elaborate instruments for laboratory use and the very simple ones intended only for use in obtaining approximate results. It embodies several novel features which I believe help to make field testing more rapid and less fatiguing to both doctor and patient.

DESCRIPTION OF THE INSTRUMENT

The arc of the perimeter, 9.2 cm., is sufficiently broad to supply an adequate background for both fixed and moving targets. The radius of the arc is 315 mm., which is a convenient working distance and yet long enough for good clinical work.

Fixation Target.—The instrument aims to make fixation steadier and more restful by supplying a variety of fixation targets. These can be interchanged by the turn of a finger, and thus several fixation targets can be provided in order to hold the patient's attention. The mechanism for this purpose consists of a small 10 mm. plane mirror set in the middle of the perimetric arc. Below the patient's chin, attached to the supporting bar, is a small housing carrying a small 10 cent electric bulb connected by means of a rheostat to the general current. In front of the bulb are two revolving disks. One disk contains an occluder and five different apertures. These are a 10 mm. round, a 5 mm. round, a cross, a triangle and a clear ring with a central black dot. The latter provides a "negative" fixation target. The other disk carries five colored patches, white, blue, yellow, red and green, each one of which can be exposed behind any one of the openings of the first disk. The patient looks into the mirror and sees the reflected fixation target. The mirror acts like a long tube and insures steady fixation, since only when the patient fixes accurately is the target visible. The targets may be changed in size, form, color or intensity by a turn of the finger. The occasional changing of the form or color of the fixation target serves to hold the patient's attention on the target much longer, and fixation then proves less fatiguing to the patient and the examiner alike.

An additional factor which insures steadiness of fixation is the fact that the fixation target is farther from the examined eye than the movable target. The reflected fixation target is about 70 cm. from the eye, while the movable target is at 31.5 cm. from the eye. It is a fact easily demonstrable that when the central fixation target is beyond the arc of the perimeter and farther than the moving peripheral target there is less tendency on the part of the patient to turn his eye from the fixation target toward the moving target.

This paper was read before the Section of Ophthalmology, New York Academy of Medicine, on March 16, 1936.

Central Color Scotomas.—Central relative scotomas can be easily detected by revolving the color disk. This brings each of the four principal colors directly into the central field without first exposing it in the paracentral field.

Moving Target.—The moving target is set in a small housing mounted on a long black handle. Here likewise there are two superimposed disks, and by a turn of the finger targets of different size and color can be presented. The sizes chosen are 1, 2, 3 and 5 mm. In addition to white, the four principal colors can be presented in these different sizes. This enables the examiner to do quantitative perimetry as well as quantitative color perimetry or a combination of the two. The targets are made self-luminous by means of a small ordinary 10 cent bulb set in the housing. This arrangement provides even illumination in all parts of

An improved perimeter-campimeter

the field. There is no diffuse illumination, but the brightness of the target can be altered to suit various conditions. Perimetry can be carried on with equal ease in a light room and in a dark room with a dark-adapted eye. For very fine work at the margin of the field instantaneous exposure can be had by means of a switch in the handle which flashes the current on and off.

Accessory Degree Numbers.—The degree numbers are shown by a scale at the back of the perimeter arc and also by a series of small perforations at the rim of the arc. This enables the examiner to see the position of the moving target from every direction and for every position of the arc. Whether the examiner works alongside, in front of, or behind the patient he can at all times see the perforated degree numbers without having to bend round the arc in order to see the scale on the back.

Campimeter.—For a study of the physiologic blindspot and of central and paracentral scotomas, a flat disk campimeter is provided. This is readily attached to the perimeter arc without disturbing or moving the patient. Thus a perimetric and campimetric examination can be carried out with a minimum of time and energy. This disk is set 25 cm. from the eye. At this distance defects of the central and paracentral field can be conveniently mapped, and yet the distance is long enough to bring out even small defects. The mapping can be made on the slate and transferred to a record chart, or a chart the duplicate of the slate may be inserted in the campimeter frame and the record filed directly.

Since writing this article my attention has been called to a description of an improved perimeter and screen by Lewkowitsch [1] of London, England. There are several features in the two instruments which are more or less similar as well as several in each of the instruments which are entirely different and unique.

1. Lewkowitsch, H.: An Improved Perimeter and Screen for Examining the Field of Vision, Brit. J. Ophth. **5**:166 (April) 1921.

Ophthalmologic Review

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FIXATION IN SCOTOMETRY

A CRITICAL INQUIRY

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The ability of the eye to fix a point of reference is essential to standard methods of scotometry. Though fixation theoretically may be accomplished by any portion of the retina, it is certainly necessary that the visual line remain (relatively or absolutely) stationary in order to permit the plotting of a defect.[1] Fixation of a point of reference on a chart by the macula constitutes the standard by which the anomalous types of fixation are judged, but macular fixation is in itself a complicated process, and many of the elements concerned are as yet poorly understood. In the following pages an effort is made to summarize certain aspects of the problem so as to emphasize the importance of this act in scotometry.

If a defect is projected on a tangent screen placed at 2,000 mm. and again on one at 330 mm., it is found by calculation that the first screen gives an enlargement of the defect that is about six times greater than that on the second screen. One would therefore think that it would be much easier to outline the defect accurately at 2,000 mm. This is doubtless so in a gross sense, but the details are lost, and in the case of a scotoma the detection of details is a most important essential. Why should detail be lost in this instance with magnification? There are perhaps a number of reasons, but the most obvious seems to lie in the fact that fixation can be maintained most perfectly at shorter ranges.

It is necessary to inquire into the mechanism of fixation in order to comprehend just what this means. Since the macula is the "organ" of fixation, its functions must be catered to. Therefore, macular function is the first step to be considered in this inquiry.

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1. Prism scotometry and scotometry in which the scotoma is caused to pass over a fixed point by directed movements of the visual line are omitted from this consideration.

MACULAR FUNCTION

The fixation spot must have a high degree of visibility (as the macula is most efficient for objects of this sort). Factors which make for visibility are (1) a high degree of reflection, (2) great amount of contrast and (3) refinement of detail. The amount of light falling on the fixation point does not of itself imply good visibility, even if the contrast between the fixation point and the background is good. The amount of light reflected back to the macula is of vital importance. In this regard, it is necessary to recall certain physiologic peculiarities of the macula, however. First is that truism stated by Helmholtz:[2] "The dimmer the light, the farther away [from the fovea] is the part of the retina that is used in looking at it." Moreover, when the fixation point is poorly illuminated, the degree of eccentric fixation is constantly changing on account of retinal adaption to the new conditions.[3] This is not an assumption without material support. The following statement is quoted from Parsons:[4] "The rate of regeneration of the substance is quicker for the cones than for the rods, which accounts for the much quicker adaption of the former (cones 3 minutes; rods 30 minutes)." The change from the mechanism of the rods to that of the cones occurs at a brightness of 0.0134 millilambert.[5] Hecht calculated that there are 572 steps in the discrimination of luminosity, and that one-third are due to rod and two-thirds to cone vision. This theory accounts for the falling off of the ability to discriminate changes in luminosity at both high and low intensities.[6]

A fixation spot loses brightness inversely as the square of the fixation distance; hence it loses much of its brightness when moved from 190 to 2,000 mm.[7] (over one-hundredth as much light). This great reduction may be so marked as to be appropriate for fixation by an eccentric

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2. Helmholtz, H.: Helmholtz's Treatise on Physiological Optics, translated and edited by J. P. C. Southall, Ithaca, N. Y., The Optical Society of America, 1924, vol. 2, p. 332.

3. Helmholtz,[2] p. 333.

4. Parsons, J. H.: An Introduction to the Theory of Perception, New York, The Macmillan Company, 1927, p. 228.

5. Luckiesh, M., and Moss, E. E.: Seeing, Baltimore, Williams & Wilkins Company, 1931, p. 63. The authors discussed the relationships of various fundamental and practical units involved in brightness. Perfect diffusion is assumed when necessary in accordance with the definitions.

6. Duke-Elder, W. S.: Textbook of Ophthalmology, London, Henry Kimpton, 1932, vol. 1, p. 897. Duke-Elder in referring to low illuminations said: "In these conditions the paracentral region is the most sensitive, being almost 1000 times more so than the fovea."

7. Comparing the stereocampimeter range with that of the tangent screen at 2 meters.

retina, which is better adapted for vision in dim light.[6] An eccentric retina, as already stated, adapts for form more slowly than the macula, so that "wandering" movements are set up, though of small amplitude.[8] These can be demonstrated by the study of after-images projected on a screen at 2,000 mm.

That the fixation point must stand out against the background with a maximum amount of contrast is obvious if maximum brightness (relative) and detail of form are to be attained. Maximum contrast is attained when a background is used which absorbs the greatest possible amount of incident light and when a fixation point is used which diffusely reflects[9] the greatest possible amount of incident light. A black background and a white point are hence to be sought, but one must keep in mind other factors having to do with the problem of general illumination.

The spot must have detail of form, as the macula is adapted to the recognition of form. The size of the fixation spot should be relatively minute, in fact approaching the threshold of the particular macular vision of the eye concerned.[10]

One may exaggerate the conditions again to make more emphatic the points discussed. The details of form of a fixation object are greatly reduced also when moved from 190 to 2,000 mm., as the size of the fixation object is not ordinarily increased when the screen is moved farther away. Thus, the ideal fixation object might be a letter from a printed page; it should probably subtend a visual angle corresponding to the subject's central (or, where this is reduced, paracentral) visual acuity, taken under the same lighting conditions as those in which the studies of the fields are made (from 7 to 20 foot candles). This would have form and would be appropriate for the macula to fix on at 190 mm., but when moved to 2,000 mm. it would become only a blurred spot.[11]

If one were to use a round button or pinhead, that principle would still pertain, for its sharpness of border, its contour and its reflexes might be recognized as giving it form at 190 or 330 mm., but it would

8. Contrast is not disturbed, as the reduction of light on the background and on the fixation point is relatively unaltered. One must keep in mind, however, that Weber's law does not hold either for very high or for very low levels (Parsons,[4] p. 186).

9. This is called diffuse reflection in contradistinction to specular reflection. The latter condition must be carefully avoided or glare results.

10. Helmholtz,[2] p. 301.

11. In actual practice this condition is inserted when studies are made on the tangent screen at 1 meter and then at 2 meters. One does not actually have occasion to move a screen from 190 to 2,000 mm.

be seen as a white blur at 2,000 mm.[12] This lack of form would render it appropriate for eccentric fixation but not for macular fixation.

The color of the fixation point probably should be white in order to enhance the contrast. The ideal fixation point would thus appear to be white, showing minute detail. On the other hand, if a dull, rather large oval ellipse were used, that isopter (of visual acuity) zone of the eccentric retina to which it corresponded would adapt for it, so that one could say that the eccentric retina was used to maintain the eye in the position of normal fixation. One concludes, then, that a chalky white letter of the alphabet, 5 minutes of arc in size, placed on a lusterless background (chart) would be an appropriate fixation point if macular function were intact.

In opposition to the ideas of a bright fixation point for the macula, it has been suggested that a bright fixation point is likely to fatigue[13] the retina easily, with the result that an after-image is formed. The retina, not yet adapted, is then shifted to one side in order to replace the image. (However, retinal fatigue, in this sense, probably does not occur.) Such a movement is of course incompatible with refined mapping.

Whether stereoscopic (binocular) fixation is used or not, it is possible to combine the central and eccentric zones for fixation by arranging lines on a chart in such a way as to give the effect of looking down a tube. An objection to this perspective appearance of the fixation spot is that it stimulates the accommodative effort. This aspect will be considered next.

INFLUENCE OF ACCOMMODATION ON FIXATION

Since recognition of the detail of near objects is normally dependent on accommodation and since satisfactory fixation depends on the maintenance of a sharp definition, it is obvious that accommodation bears an important relation to fixation. Near vision for the recognition of detail is a provision of nature.

Helmholtz,[14] in discussing the form sense, recalled a significant fact:

> The resolving power is much less in the lateral parts of the retina than in the yellow spot; the decrease near the center of the retina being less than it is farther away from it. . . . Another noteworthy result from these measurements is that

12. To be sure, under ordinary conditions (when the eye is adjusted for infinity), the blur circle for 330 mm. is about 0.1 mm. larger than one for a distance of 2,000 mm. (Helmholtz,[2] vol. 1, p. 132), but it will be shown in the following paragraphs how the blur circle for near vision may be reduced to the proportions of one at infinity.

13. There is a growing tendency to oppose the idea of retinal fatigue (Duke-Elder,[6] p. 968; Parsons,[4] p. 180).

14. Helmholtz,[2] p. 34.

in accommodation for distance, the falling off of the resolving power toward the periphery of the retina seems to proceed more rapidly than it does in near vision.

Tscherning [15] gave definite data on this point.

Prolonged maintenance of steady accommodation while a nearby stationary object is being fixed seems to be an unusual combination, irrespective of the distance of fixation. If the accommodation can be relaxed entirely, fixation seems easier; in fact, when one is attempting to fix a point on a tangent screen, one will often become conscious that the eye is entirely out of focus. (While this may be detrimental to accurate fixation, it does not interfere with reasonably accurate plotting of eccentrically located defects.) I have had a + 10 diopter lens made with the center (2 mm. in area) ground plane. As fixation through the center is sharp, eccentrically located defects are as readily plottable (though slightly enlarged) as if no such lens were before the eye. This type of lens steadies fixation, because if the subject attempts to look at the object directly, he is at once reminded of the act by the blurring of the +10 diopter part of the lens. The ideal condition seems to be to fix the point clearly at the reading distance yet not to exert the faculty of accommodation.[16] Fortunately this can be accomplished fairly easily by the use of the appropriate collimating lens (fig. 1).

Light reflected from a fixation point follows a divergent course. If the screen is placed 2,000 mm. from the subject, these rays can be rendered parallel before they enter the patient's eye by placing a + 0.50 diopter lens before the eye.[17] As it is unnecessary for the eye to accommodate for parallel rays, the fixation point may be seen without this exertion. A + 1.00 diopter lens would be placed in the same way for a screen at 1,000 mm.; a + 3.25 diopter lens, at 330 mm., and a + 5.25 diopter lens, at 190 mm. The screen is thus placed optically at infinity, but it is actually at accommodative close range. The details are, moreover, slightly enlarged (at first) by this collimating lens, and the subject seems to be able to respond more definitely when the object disappears and reappears at the borders of a defect. The sharper details being more appropriate to the macular functions, fixa-

15. Tscherning, M.: Physiological Optics, ed. 4, translated by C. Weiland, Philadelphia, Keystone Publishing Co., 1924, p. 209.

16. Nagel (in Zoethout, W. D.: Physiological Optics, Chicago, Professional Press, 1927, p. 152) made this interesting statement: "With characters of the same apparent size, small characters nearer the eye were easier to discover than larger characters farther away." He said that the reason for this is difficult to explain and suggested the possible influence of accommodation.

17. Rays of light arising from a point 1 meter away are rendered parallel by a +1 diopter lens. A point at half this distance would require a +2 diopter lens to render the rays parallel. It is thus simple to calculate the lens which will collimate the divergent rays from a chart at any desired distance.

tion is encouraged. When the screen is placed at 190 mm. (as in the stereocampimeter), there is linear magnification (by the collimating lens) of about 15 per cent.[18]

When a scotoma is successfully plotted at different ranges, not only is there an increase in the size of the defect, but the defect becomes "thinner." If a defect is plotted at 6 meters instead of at 2 meters, it is more easily appreciated. The borders are less definite, on account of the increased influence of poor fixation, but the retina projected on the infinitely removed sphere of the visual field (at 6 meters) is spread over so great an area that patients often describe haziness for far vision but absence of objects for near vision (within the area of the scotoma). Hence this increase in the loss of definition of a defect shows a relation to the distance of fixation. If this same principle is carried further, it is found that a bit of black paper pasted on the lens of an eyeglass produces only a relative scotoma, and the same is true of corneal

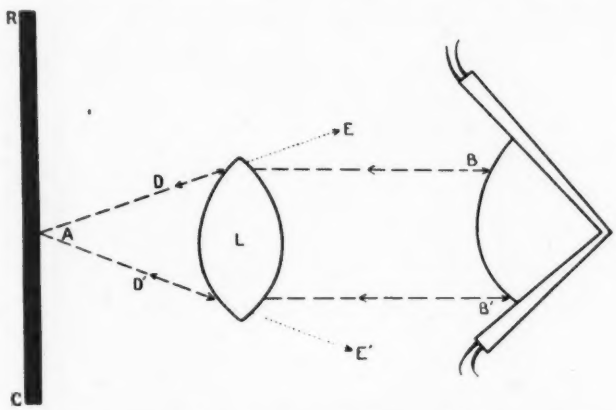

Fig. 1.—The collimating lens. *RC* represents the tangent screen and *A* the fixation point. Lines *A D E* and *A D' E'* show divergent course of light from the surface of the tangent screen. Divergent rays *A D* and *A D'*, impinging on lens *L*, are rendered parallel (*A D B* and *A D' B'*), so that they enter the eye as if from infinity.

opacities unless very minute test objects are used. Vitreous opacities, however, become so positive as to appear densely black to the patient.

The collimating lens has, in effect, the power of contracting the visual field, thus compressing, as it were, the relative defects into positive ones.

18. This figure is obtained by placing a —5.25 diopter lens over the campimeter lens of one side, thus neutralizing the +5.25 diopter lens in the instrument. A green glass is placed before the same eye. The prisms of the instrument are then adjusted until the green-appearing chart overlaps the opposite chart and the difference in size can be measured by counting the degree squares (smaller squares). (The central partition on my stereocampimenter is removable.) It is found that 20 degrees of the larger are occupied by 17 degrees of the smaller (Green).

MUSCULODYNAMICS OF FIXATION [19]

It is obvious that the anatomic position of rest would not be appropriate for fixation, because in total ophthalmoplegia externa and immediately after death, when the anatomic position of rest may be said to occur, the eyes rarely look straight ahead but usually diverge or occasionally converge. Should such a position be desirable, there is no known way of maintaining it. The extra-ocular muscles maintain a normal tonus at all times. When the eyes are uninfluenced by stimuli for orientation (accommodation, muscle balance, etc.) they assume what may be called the physiologic position of rest. It represents a balance of muscle tonus of the extra-ocular muscles. Such a state of affairs occurs only when both eyes are looking at the same point in infinity, and this occasion must arise rarely, unless artificial conditions are created to accomplish it (as with the stereocampimeter). This position, even when it does occur, being dependent on a perfect balance of muscular interaction, theoretically could not be stationary, because of the inherent irregularities of muscle contraction [20] during the maintenance of normal tonus.

Fixation, as it is commonly known, particularly for points inside infinity, has the result of an unequal distribution of work among the extra-ocular muscles, so that one has the right to expect the insertion of other factors further disturbing the theoretically perfect fixation. The actual movements of the eye during fixation have been studied elaborately by many workers and by many methods, and there is a surprising unanimity in the conclusions. It seems to be agreed that the efforts of the extra-ocular muscles to maintain perfect fixation results in minute jerky movements of the eyes,[21] which occur in two components:

1. Relatively large jerky movements through an angle of 4 minutes of arc occur at intervals of from one to two and one-half seconds (fig. 2).

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19. (*a*) Duke-Elder,[6] p. 579. (*b*) Adler, F. H., and Fliegelman, M.: Influence of Fixation on the Visual Acuity, Arch. Ophth. **12**:475 (Oct.) 1934. I use Duke-Elder's figures in the following discussion because he apparently drew his conclusions from the reports of a great many workers. This seems safer than to accept the data of a single worker.

20. The simplest evidence of this phenomenon is the muscle sounds, which are easily elicited if an appropriately formed stethoscope is applied over the "belly of the extra-ocular muscles."

21. While it is not possible to cite actual evidence, it seems obvious that binocular fixation may modify the fixation movements to such an extent as to warrant the statement that monocular fixation is to all intents and purposes initiated by an entirely different mechanism than is binocular fixation.

2. During the period of rest there are fine jerky movements which have an amplitude of 1 minute of arc and which last for from one to two and one-half seconds. There is also a minute variation in the position of the head.

Adler and Fliegelman [19b] concluded from a carefully controlled series of studies that there are three components: (1) rapid shifts occurring once every second, with an amplitude of 30 minutes of arc; (2) waves, occurring once every two seconds, with an amplitude of 3 minutes and

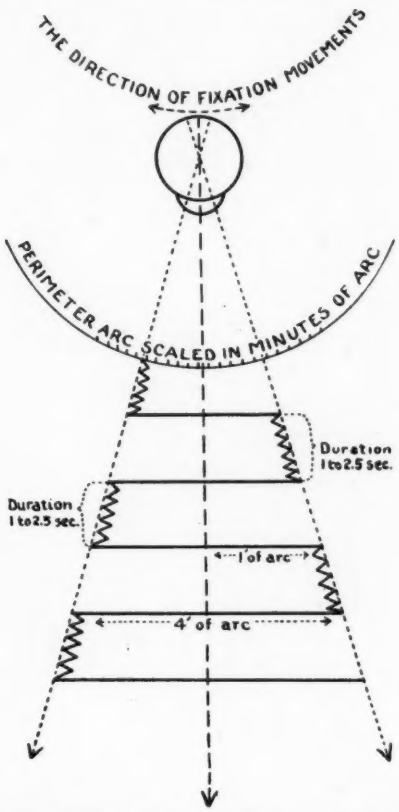
THE DIRECTION OF FIXATION MOVEMENTS
PERIMETER ARC SCALED IN MINUTES OF ARC
Duration 1 to 2.5 sec.
Duration 1 to 2.5 sec.
1' of arc
4' of arc

Fig. 2.—Schematic representation of the movements of the eye during fixation.

1 lambert = 0.3183 candle per square centimeter = 2.054 candles per square inch.
1 candle per square centimeter = 3.1416 lamberts.
1 candle per square inch = 0.4868 lambert = 486.8 millilamberts.
1 millilambert = 0.001 lambert.
1 foot lambert = 1.076 millilamberts.
1 spherical candle emits 12.57 lumens.
1 foot candle = 1 lumen incident per square foot.
1 lux = 1 lumen incident per square meter.
1 lambert = 1 lumen emitted per square centimeter.
1 lumen emitted per square foot = 1.076 millilamberts.
1 foot lambert = 1 lumen emitted per square foot.
1 millilambert = 0.929 lumen emitted per square foot.

43.5 seconds of arc, and (3) fine vibratory movements lasting for one one-hundredth or two one-hundredths second, with an amplitude of 2 minutes and 14 seconds of arc. In other words, once every second the eye makes a readjustment which carries the image over twenty-six cones. Furthermore, when the eye is steadiest, a point image is moving across the retina an angular distance of over 2 minutes, and even then it maintains one position for not longer than one-fifth second.

This means that the actual area of retina used for fixation is constantly shifted about over a relatively large arc. The amplitude of these movements is apparently the same whether fixation is for far or for near vision. They bear no relation to the pulse or the respiratory rate, and there is marked individual variation in the rate and duration. The movement is in general horizontal, but there is a rotary element in all probability so that the path of a retinal point will be an irregular ellipse.

Besides these fixation movements, there are movements associated with winking: (1) recession of the globe of about 1 mm.[22] and (2) upward turning of the globe of about 15 degrees.[23] An explanation of the origin of these movements is not of interest here, as it is of no concern whether they occur as a part of the mechanism for the maintenance of retinal activity in the area of image formation or whether they arise entirely within the neuromuscular mechanism without the influence of sensory stimuli. Graphically, these movements may be depicted as in the accompanying illustration (fig. 2).[24]

It is obvious that such movements result in the passage of an image over a minute area of the retina, for instance, with the movement of 4 minutes of arc[25] the image passes over about 0.02 mm. of retina (the movement of 1 minute of arc covers about 0.005 mm. on the retina). If a long pointer could be attached to the visual line so that it would move with these oscillations of the globe, it would traverse an appreciable distance on the tangent surface instead of remaining steadily in one place.[26] Calculations to show the extent of this area from the

22. Duke-Elder,[6] p. 581.

23. Miles, W. R.: Elevation of the Eyeballs on Winking, J. Exper. Psychol. **14**:311 (Aug.) 1931.

24. Luckiesh, M.: Seeing and Human Welfare, Baltimore, Williams & Wilkins, 1934, p. 96. The author said: "The average stopping-time of the eye is .17 second. In extensive tests of this it was found that the briefest interval of time that it is possible for an observer to fixate an object in order to gain an adequate visual impression varied for the most part from .07 to .30 second. The average was .17 second."

25. Taking the distance from the second nodal point to the retina as 17.055 mm. and multiplying it by the natural tangent value for 4 minutes of arc (or for 1 minute of arc).

26. The natural tangent value for 4 minutes of arc is 0.001164 mm.; for 1 minute it is 0.000291 mm.

various distances of fixation, as met with in standard instruments, give the following results:[27]

Distance of Fixation, Mm.	Minutes of Arc	Tangent Plane, Distance in Mm.
	Coarse Movement of Fixation	
190	4	0.22116
330	4	0.38412
750	4	0.87300
1,000	4	1.16400
2,000	4	2.32800
	Fine Movement of Fixation	
190	1	0.05529
330	1	0.09603
750	1	0.21825
1,000	1	0.29100
2,000	1	0.58200

In other words, if a pointer could be attached to the eye, 4 minute movements would cause it to oscillate on a tangent screen placed at a distance of 2,000 mm., a distance of 2.328 mm.; at 1,000 mm., 1.164 mm.; at 330 mm., 0.384 mm.; at 190 mm., 0.221 mm. It thus becomes obvious that a minute scotoma as projected by the retinal defect is not stationary on the tangent screen and that the more remote the screen, the greater the area covered by the shiftings. The borders are thus much more difficult to plot and in fact may be missed entirely unless all other conditions are particularly favorable.

From these figures it might seem impossible to discover a scotoma as small as one equal to 4 minutes of arc, because the movements of the eye would carry it rapidly back and forth and so bring the image of the object on a seeing portion of the retina. This is not actually so, for it is easy to detect a scotoma which has reduced vision from 20/20 to 20/30. Such a defect can apparently be appreciated during the fine fixation movements, however.[28] In spite of these movements, Luckiesh[24] has demonstrated that the eye can gain adequate visual impressions by fixating for 0.17 second; yet the finer movements are going on during that time.

Added to the actual movements, however, are other factors which, theoretically, distort the scotoma. These are retinal light scatter,

27. It must be realized that these figures represent the projection of minutes of arc on a tangent surface.

28. If the shape of the letters cannot be distinguished in the 20/20 line but can be in the line of larger letters, there is a defect in retinal function equal to the difference in these two letters. The diameter of the limb of a letter in the 20/20 line is 1.77 mm., while that of one in the 20/30 line is 2.65 mm.; hence the difference is 0.88 mm. The fine fixation movements (at 6 meters) cover 0.29 mm.; hence they would occur entirely within the defect. The coarse movements cover 1.16 mm.

spherical and chromatic aberration of the refractive media, other optical defects of the eye and probably minute discrepancies of the conducting and registering neuromechanism. These factors make it evident that the geometric relations of the functionally depressed retina and those of the projected defect (scotoma) cannot be stated in precise mathematical terms. The average width of the angioscotoma near the blind-spot of Mariotte is 1 degree. If this is projected at various distances on the tangent screen it is found that at 2,000 mm. it is 34.9 mm. wide; at 1,000 mm., 17.4 mm. wide; at 330 mm., 5.7 mm. wide; at 190 mm., 3.3 mm. wide.

When attempts are made to map a scotoma at 1,000 or 2,000 mm. ranges and the examiner finds that the object seems to fade from view in relatively large areas, he must realize that this is due not to "retinal fatigue" but to the indefiniteness of the border of a scotoma from 18 to 35 mm. wide. There is no sharp border to such shadows; they have a sort of penumbra.[29] Moreover, one would not expect a retinal lesion of measurable proportions to result in the production of a scotoma of predictable relative size, at least in a geometric sense. The fact remains that, in spite of these discrepancies, minute defects can be mapped, just as small print can be read, and one may be justified in assuming that this is possible by means of a sort of scanning, like search-lights sweeping across the night skies in search of an airplane. The recognition of the border of a scotoma, then, is akin to the recognition of the alinement of two hairs; the recognition is the result of the averaging of a group of stimuli rather than the product of a single fleeting impression.[30]

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29. Bailliart, P.: The Retinal Circulation in the Normal and Pathological States, translated by J. E. Lebensohn, Chicago, Professional Press, Inc., 1928, p. 16. Bailliart said that the retinal arteries as they emerge from the disk are 210 microns (0.21 mm.) in diameter and the veins are 245 microns (0.245 mm.) in diameter.

30. The following quotation (Adler, F. H.: Clinical Physiology of the Eye, New York, The Macmillan Company, 1933, p. 136) will serve to emphasize this point: "These movements must not be looked upon as defects which the individual tries to overcome in order to see acutely. As a matter of fact, if absolute fixation could be obtained it would be a decided handicap to vision because of the rapid sensory adaption shown by the retina. In addition to adaption, the retina would suffer still further from accurate fixation by the development of troublesome after-images. As a final objection to any theory which assumes that different portions of a zone can transmit different degrees of intensity over the single connecting fiber, it may be said that all recent work on sensory receptors has shown that they obey the all-or-none law similar to the nerve impulse in a motor fiber. There is every reason therefore to believe that fractional stimulation of a cone or even fractional conduction in a nerve fiber, is impossible.

Now it is a well known fact that when one is dealing with a large number of measurements, the accuracy obtained by averaging a large number of these, is

Thus far the problems of fixation have been discussed as they affect one eye. When some form of stereoscope is used (such as the stereocampimeter or screen of Goldman), not only do the movements of fixation of each eye need consideration but also the fact that the eyes will not maintain an absolutely constant angle between the respective visual lines (optical or actual). This is commonly noted when the routine tests for muscle balance are made (Maddox rod, red glass and screen and parallax tests) for near as well as for far vision. The swaying of the visual lines is so marked that the patient will remark about positional variation of the images without prompting. If an actual central scotoma exists, there is a deviation of the affected eye which brings the next most efficient area of seeing retina into line for fixation. This occurs most markedly when the unaffected eye is prevented from fixing. The stereoscope permits the unaffected eye to fix and yet encourages the affected eye to "aim" as if it were fixing normally. There are two exceptions to this: First, if the defect has existed for a number of years or was acquired in infancy, the affected eye cannot take up its normal position, in spite of the demand of the unaffected eye (as divergence, and rarely convergence, of blind or nearly blind eyes). Second, if there is a marked departure from normal muscle balance, prismatic correction must be made to assist the proper alinement. (In my experience this has seldom been satisfactorily accomplished, as the swaying movements are greatly exaggerated by the very mechanism which accounts for the great muscular imbalance and also by the loss of macular vision.)

With these points in mind, it thus becomes obvious that if one eye is occluded during a stereocampimeter examination, one variable and less controllable factor will be eliminated as a source of error. It is

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actually greater, even though the physical measurement is done roughly, than if a few measurements were done with extreme care. In the same manner the shift in position of a linear image on the retina by stimulating a relatively large number of receptor units, each having its own local sign, may result in more accurate localization in consciousness of the line than can be accounted for by its shift in position from one cone to another. The length of the line according to this theory will materially determine the accuracy with which a shift in position can be visualized, since the longer it is, the more cone groups will be affected. Experiment shows this to be so. Within certain limits the alining power of the eye is considerably enhanced by increasing the length of the line."

As already noted, outlining the scotoma, as the examination progresses, is akin to calling forth the alining power of the eye. There is evidence of an effort of the eye to use the borders of the plotted defect as a secondary means of fixation. In fact, as these studies have progressed—using the minute object as a test—the conviction has been impressed on me with increasing force that the alining power of the eye is more responsible for the successful outlining of the defect than has been previously suspected.

only occasionally that "binocular fixation" (perhaps "conjugate fixation" would be a better term, as it indicates a yoking of one eye with the other during the act of fixation) seems to give better results in such cases if there is a very small central scotoma in one eye.

In the study of a small series of cases [31] in which central retinitis of each eye produced a central scotoma in each field, the relative position of the fixation lines was the same as in normal eyes, apparently because these eyes were turned so that the acuity isopters of equal value were used for fixation instead of the macula. This is more likely to be so if the central scotomas are equal in size and degree (as they were in these cases). (As a rule one does not expect in this type of case to see deviation of the eyes long after the onset of the condition, but one does expect to if a central scotoma affects one eye only.)

31. There were only six cases (a rather insufficient group) for this study on account of the rarity of satisfactory conditions: (1) an atrophic stage of bilateral central chorioretinitis (2) which had developed in an adult after the full development of acuity, fixation and fusion, (3) without a previous demonstrable muscular anomaly or (4) nystagmus, (5) equal-sized atrophic lesions in the two eyes (measured with the graticule of a Keeler scope); equal visual acuity in the two eyes, and (6) satisfactory cooperation for mapping. In case 1 vision without correction in the right and in the left eye, respectively, was 6/15 +3 and 6/18; in case 2, 6/15 +1 and 6/18; in case 3, 6/15 +2 and 6/18; in case 4, 6/18 and 6/18; in case 5, 3/21 +2 and 3/21, and in case 6, 3/21 +2 and 3/21.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

UNIVERSITY NEWS

Dr. Don Marshall has been appointed full time assistant professor of ophthalmology at the University of Michigan Medical School.

SOCIETY NEWS

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat.—The following officers were elected for the coming year at the fourteenth annual meeting of the Section on Eye, Ear, Nose and Throat of the Connecticut State Medical Society, held at Hartford on May 21: Dr. Walter L. Hogan, Hartford, chairman; Dr. Charles T. Flynn, New Haven, vice chairman; Dr. Shirley H. Baron, New London, secretary-treasurer. Dr. Conrad Berens, of New York, addressed the section on "Clinical Considerations of Ocular Fatigue," and Dr. E. Ross Faulkner, of New York, spoke on "Review of Operative Technic in Nose and Throat Surgery."

North Dakota Academy of Ophthalmology and Oto-Laryngology.—At a recent meeting of the North Dakota Academy of Ophthalmology and Oto-Laryngology, held at Jamestown, the following officers were elected: Dr. J. P. Miller, Grand Forks, president; Dr. H. Rosenberger, Bismarck, vice president; Dr. F. L. Wicks, Valley City, secretary-treasurer. Dr. Erling W. Hansen, Minneapolis, visiting clinician, spoke at the meeting on "Allergy in Ophthalmology." Other speakers were: Dr. G. A. Larson, Fargo; Dr. G. M. Constans, Bismarck, and Dr. N. A. Youngs, Grand Forks.

GENERAL NEWS

Fifteenth International Ophthalmological Congress.—The Fifteenth International Ophthalmological Congress will be held in Cairo, Egypt, from Dec. 8 to 14, 1937. The two official subjects of the congress will be: "Arterial Hypertension of the Retina" and "Endocrinology and the Eye." Papers to be read in the scientific meeting should be sent directly to Dr. Marx, Oostzeedijk 316, Rotterdam. A subscription fee of 50 Swiss francs and requests for further information should be sent to the Secretary General, XV. Concilium Ophthalmologicum, P.O.B. No. 2001, Cairo, Egypt.

Summer Graduate Course in Ophthalmology, University of Rochester.—The department of ophthalmology of the University of Rochester School of Medicine and Dentistry will conduct the seventh annual summer graduate course of lectures and demonstrations in ophthalmology from July 27 to 31, 1936. The course is designed to offer a series of lectures and demonstrations of interest to all practi-

tioners in ophthalmology. Recent advances and observations in ophthalmology, as related to general medicine and surgery, will be stressed.

Ophthalmologic surgery, ophthalmoscopy and medical ophthalmology, perimetry and ophthalmic neurology, slit lamp microscopy, ophthalmic myology, pathology, retinoscopy and refraction will be covered by this course. The tuition fee for the course is $40. Further information may be secured from Dr. John F. Gipner, Strong Memorial Hospital, Rochester, N. Y.

Course in Ophthalmology, New York Post-Graduate Medical School.—A course in "Motor Anomalies of the Eye, for Ophthalmologists" will be given by Dr. James W. White at the New York Post-Graduate Medical School, Columbia University, from July 13 to 19, 1936. Information regarding application and the registration fee may be secured from the director, 303 East Twentieth Street, New York.

Lucien Howe Prize.—Dr. Arthur J. Bedell, of Albany, N. Y., was awarded the Lucien Howe Prize by the Medical Society of the State of New York at its meeting in New York on April 28, 1936.

Obituaries

HENRY H. TYSON, M.D.
1865–1936

Dr. Henry H. Tyson, son of Henry H. and Leticia Tyson, died suddenly of heart disease at his home on April 18, 1936, at the age of 71.

Dr. Tyson was educated at the College of the City of New York and was graduated from the New York University Medical College in 1887. Immediately after graduation, he became associated with the ophthalmic department of the New York Ophthalmic and Aural Institute, where he worked under Dr. Herman Knapp. He continued his association with this same institution after it became the Herman Knapp Memorial Eye Hospital in 1913. In 1895 he was appointed surgeon, and he continued in that capacity until his death. He was also a clinical instructor in diseases of the eye at the College of Physicians and Surgeons from 1903 to 1915.

In addition to designing a tattooing needle which was used in the application of color to the cornea, Dr. Tyson was the discoverer of the Tyson-Clarke ocular syndrome in dementia praecox. He was one of the first to discover the relationship between tuberculosis and keratitis disciformis.

With Dr. M. J. Schoenberg, he presented before the American Medical Association in 1914 an exhaustive study on experimental researches on the inhalation of methyl alcohol. While Tyson did not contribute extensively to the ophthalmic literature, his papers were always well considered and practical, and his discussions were interesting and sound.

During the World War Dr. Tyson was special ophthalmic examiner for the third district of the New York Draft Board. He was ophthalmologist to the Sea-Breeze Hospital, in Brooklyn, and to Letchworth Village, Thiells, N. Y.

He was a fellow of the New York Academy of Medicine, and a member of the county and state medical societies, the American College of Surgeons and the American Ophthalmological Society. He joined the American Ophthalmological Society in 1906 and was one of its oldest members. He was also a member of the Medical Veterans of the World War, the Oxford Ophthalmological Congress and the New York Athletic Club.

He was a devoted member of the New York Ophthalmological Society for thirty years and rarely missed a meeting. He was president

of that society for one year and chairman of the Section of Ophthalmology of the New York Academy of Medicine for two terms.

Dr. Tyson had a long and successful career in the practice of ophthalmology and was greatly interested in the subject at all times. His sudden death came as a distinct shock to a host of medical friends and admirers. Although of a retiring disposition, he had by years of earnest and kindly living endeared himself to many friends both in and out of the medical profession, so that his passing is a great loss to the profession, to the community and to the hospital with which he had been connected for so many years.

Surviving him are two children, Mrs. Eleanor Tyson Bridgman and Henry B. Tyson, and several grandchildren.

GEORGE HUSTON BELL.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE STRUCTURE OF THE SCLERAL CANALS AND THEIR RELATIONS TO THE VENAE VORTICOSAE AT VARIOUS AGES. E. MARCHESINI, Ann. di ottal. e clin. ocul. **63:** 689 (Sept.) 1935.

Twelve eyes with posterior segments normal except for the changes due to age were examined with especial reference to possible changes in the sclera with age. Two were from young adults aged 20 and 28, two from persons aged 51 and 59, and eight from persons aged from 60 to 87. Certain changes in the eyes were found, chiefly in persons past 60, and more advanced changes in those of greater age. These consisted in a greater compactness of the scleral structure, the fibers becoming thinner, more parallel and more closely opposed to each other, and a reduction in number and size of the fixed scleral cells. The sclera about the vortex veins became more dense, and in three eyes of persons of 70, 75 and 87 a special condensation of the sclera was seen about the veins, forming a sort of sclerotic cuff. The connective tissue, which in young eyes was arranged loosely about the veins so as to leave available a certain perivascular space for expansion, was much thinner or entirely absent in the older eyes, with a few exceptions. In only one eye was definite arteriosclerotic thickening of the venous wall observed. The findings confirm those of Weichselbaum and support the possibility that such changes, by removing a mechanism which allows the veins to expand when the intra-ocular tension is increased, may play a part in glaucoma.

S. R. GIFFORD.

Bacteriology and Serology

A STUDY OF THE PNEUMOCOCCUS GROUP FROM THE INFLAMED CONJUNCTIVA AND LACRIMAL SAC. S. H. MCKEE, Am. J. Ophth. **18:** 1021 (Nov.) 1935.

This article does not lend itself to abstracting. It is based on the possibility that local and general serum treatment of patients with pneumococcic infections of the various types may lead to the establishment of a new and thorough medication.

W. S. REESE.

THE COLON BACILLUS IN OCULAR PATHOLOGY. J. FRANÇOIS, Arch. d'opht. **52:** 424 (June) 1935.

Two cases of infection with the colon bacillus are described: one, a case of metastatic panophthalmitis; the other, of congenital dacryocystitis. Cultures were obtained and experiments made on rabbits. The conditions which have been found due to this organism are listed and briefly discussed. The results of the experiments conform to those

reported by other workers. The conclusion reached from clinical observation, confirmed by laboratory study, is that under certain conditions the colon bacillus is able to infect the eye directly and by metastasis. Its presence in the eye is exceptional.

S. B. MARLOW.

BIOLOGIC AND EPIDEMIOLOGICAL STUDIES ON STREPTOCOCCI OF THE EYE. S. SPIRATOS, Arch. d'opht. **52:** 505 (July) 1935.

Spiratos describes a case of metastatic ophthalmitis following streptococcic pneumonia, which he encountered during a study of many types of streptococcic inflammation of the eye. He could find reports of only four instances of a similar condition in the literature. In his case Streptococcus viridans was found in the vitreous and in the sputum, while Streptococcus haemolyticus was found in the conjunctival secretion. Culturing the blood gave no growth, probably because of the late stage at which it was done. In seeking an explanation for the occurrence of these two organisms in this case the question of transformation arises. This is discussed at some length, Spiratos leaning to the opinion that it is a better explanation in this instance than the coincidence of two different organisms. The occurrence of Str. haemolyticus, according to the observations made, is greatest during December and January. He points out that this is of special significance in relation to the surgical work done during this period.

S. B. MARLOW.

Biochemistry

SODIUM CONTENT OF AQUEOUS, VITREOUS AND SERUM: COMPARATIVE STUDY ON OXEN. P. W. SALIT, Am. J. Ophth. **18:** 706 (Aug.) 1935.

Salit found the sodium contents of aqueous and vitreous to be identical and that of serum approximately 9 per cent greater.

W. S. REESE.

THE BLOOD-AQUEOUS BARRIER AND VITAMIN C: I. THE PERMEABILITY OF THE BLOOD-AQUEOUS BARRIER AND THE ASCORBIC ACID CONCENTRATION IN THE ANTERIOR CHAMBER. H. GOLDMANN and W. BUSCHKE, Arch. f. Augenh. **109:** 205, 1935.

Large amounts of ascorbic acid (the reduced form of vitamin C) have been found in the normal aqueous of man and animals. Its concentration in rabbit aqueous is about 20 mg. per hundred cubic centimeters. The vitamin C in the aqueous comes, for the most part, from the metabolism of the lens. The lens is known to reduce the oxidized form of vitamin C which enters the anterior chamber from the blood stream; but, in addition to this, the lens itself probably forms small amounts of the vitamin. As a result the concentration of the vitamin in the aqueous is about ten times that in the blood stream. It is known that vitamin C can diffuse freely from the blood into the aqueous, but the high concentration found in the aqueous suggests the possibility that this permeability is not reversible and that the aqueous-blood barrier

normally prevents vitamin C from moving back into the blood stream from the aqueous.

The present experiments were designed to test this hypothesis. Rabbits were used throughout the experiments. The concentration of vitamin C in the aqueous was determined by Harris' method. Special pipets were used to withdraw the aqueous from the anterior chamber so that it was never in contact with air except in the pipet itself. A preliminary series of experiments proved the concentrations of vitamin C equal in the two eyes in a series of normal rabbits. The permeability of the blood-aqueous barrier was increased by injecting sodium chloride subconjunctivally in concentrations of from 5 to 15 per cent. An increase in the concentration of protein in the aqueous and a more rapid appearance of fluorescein in the anterior chamber indicate to the authors that the injection of hypertonic solutions of sodium chloride effectively increases the permeability of the blood-aqueous barrier. They found a reduction in the vitamin C content of the aqueous in the majority of cases in which the hypertonic salt solutions were employed. This reduction in the concentration of vitamin C did not run parallel with the increase in the protein content of the aqueous but followed more nearly the increase in the fluorescein in the anterior chamber. They regard the latter as a better indicator of permeability than the increase in the concentration of protein. Injections of epinephrine into a normal eye did not raise the concentration of vitamin C in the aqueous but, on the contrary, sometimes lowered it. Intramuscular injections of theophylline also lowered the concentration of vitamin C in the aqueous.

Goldmann and Buschke conclude that the blood-aqueous barrier normally prevents the reabsorption of vitamin C from the aqueous into the blood but that when the permeability of this barrier is increased vitamin C is rapidly reabsorbed into the blood stream. They discuss the possible relation which this may have in the production of cataract, more particularly cataracta complicata, in which they believe the inflammatory process (uveitis) may so increase the permeability of the blood-aqueous barrier that the vitamin C necessary for the metabolism of the lens disappears from the aqueous and the lens becomes opaque.

F. H. Adler.

Conjunctiva

The Reticulum in the Chronically Hyperplastic Conjunctiva. H. D. Lamb, Am. J. Ophth. **18:** 724 (Aug.) 1935.

Lamb found fairly uniform changes as regards reticulum in the hyperplastic conjunctiva in cases of trachoma with the exception of those involving the cornea. Comparing his observations with those of Beigelman on folliculosis, he concludes that it is difficult to see how the demonstration of reticulum in the hyperplastic conjunctiva offers any assistance in the early diagnosis of trachoma. Similar changes were also seen in a case of chronic lymphatic leukemia.

W. S. Reese.

The Conjunctivitis of Scarlet Fever. H. Otto, München. med. Wchnschr. **82:** 1987 (Dec. 13) 1935.

The majority of the infectious diseases begin with conjunctivitis. The teaching in the past has been that tearing, photophobia and redness

of the conjunctiva are not a part of scarlet fever. Otto's experience has been otherwise. In the Halle-Wittenberg clinic during the years from 1927 to 1935, more or less definite conjunctivitis was noted in 52, or 5.8 per cent, of the 891 cases of scarlet fever. Otto divides the types as follows:

I. Bilateral conjunctivitis
   1. Conjunctivitis of the bulb with fine injection of the superficial blood vessels without photophobia, tearing or heaviness
   2. Conjunctivitis of the bulb with violent diffuse injection of blood vessels
   3. Diffuse fiery-red inflammation of the bulb with and without photophobia, tearing and heaviness
   4. Forms 1 to 3 with more or less marked edema
   5. Tarsal and bulbar conjunctivitis of grades 1, 2 and 3 with and without photophobia and tearing but with blepharospasm
   6. Blepharoconjunctivitis
   7. Pure blepharitis with and without marked edema
   8. Angular conjunctivitis—medial
   9. Angular conjunctivitis—lateral
   10. Angular conjunctivitis with more irregular situation
   11. Mild conjunctivitis which later becomes marked and also returns periodically

II. Unilateral conjunctivitis
   1. The forms classified under I may affect only one eye
   2. Unilateral conjunctivitis which later involves the other side

III. Complications
   1. Perforation of the globe with prolapse of the iris, fiery-red injection of the tarsus and bulb with blepharitis and edema of the lid
   2. Secondary corneal inflammations
   3. Phlyctenules of the cornea in connection with those of the conjunctiva

The early conjunctivitis presents itself from the first to the sixth day of the disease, usually together with lesions of the skin and occasionally before these. It remains for from two to sixteen days. The late conjunctivitis occurs between the fifteenth and the fiftieth day and lasts from eight to twenty-five days. In only four cases of scarlet fever seen by Otto was rheumatism present with conjunctivitis. Because of the finding of an increase in the number of eosinophils in the blood there is some reason to believe that the conjunctivitis of scarlet fever may be an allergic reaction. It also may be due to the toxins of the organism causing the scarlet fever. The hemolytic streptococcus is found in the conjunctival sac no more frequently than other organisms, and therefore the possibility of a direct infection or of one due to transference through the tear passages is not to be considered.

L. L. MAYER.

Cornea and Sclera

"PALM LEAF" OPACITIES OF THE CORNEA: RESIDUALS OF INTERSTITIAL KERATITIS (ANNULAR TYPE OF VOSSIUS). P. BONNET, Arch. d'opht. **52:** 625 (Sept.) 1935.

The disposition of these opacities in the deeper layers of the cornea produces a figure which is best compared to a palm leaf. They are encountered rather frequently and have been known for some time, illustrations of them having appeared in the first edition of A. Vogt's "Atlas of the Slitlamp-Microscopy of the Living Eye" and in a paper by Gallemaerts. However, Bonnet suggests that these opacities have not been sufficiently individualized as residua of interstitial keratitis. He decribes the opacities in some detail and with extensive illustrations. They are seen only on examination with a slit lamp. They are present on both sides and are more frequently seen in elderly women, although he has observed their development in a 13 year old girl with active interstitial keratitis. Although it is possible that they may occur in other forms of deep keratitis, they are especially significant signs of the occurrence of interstitial keratitis many years before. They constitute, so far as present knowledge permits, an indelible stigma of hereditary syphilis.

S. B. MARLOW.

THE SYNDROME OF BLUE SCLERAS. C. CORDERO, Arch. di ottal. **42:** 521 (Dec.) 1935.

This concludes an article previously reviewed in part, which constitutes a complete monograph on the subject of blue scleras. Cordero here discusses certain phenomena associated with the condition aside from the well known fragilitas ossium. Deafness, the third symptom in the syndrome described by van der Hoeve, Cordero noted in two of his patients, and in the others, abnormalities of the vestibular mechanism, usually hyperirritability. Only one showed the blue discoloration of the tympanic membrane which has been described in a few cases recorded in the literature. Syndesmolysis, or frequent dislocation of various joints, was present in all his cases. Neuromuscular changes were present in all, consisting in hyperirritability as shown by electric tests. None of the patients showed psychic abnormalities. One showed marked diminution in the elastic tissue of the skin. Two patients showed anomalies of the teeth, one having lost all the teeth at an early age, and all gave a history of late eruption of the teeth. Fragility of the nails and loss of hair were observed in most patients. The blood calcium content was normal, as were other findings of the blood chemistry except that cholesterol was increased in all but one of the patients, reaching 400 mg. in two. The dominant hereditary character of the syndrome was evident in Cordero's family, seventeen of thirty-eight members being affected. After a review of various theories, he gives his reasons for believing that hypoplasia of both mesenchymal and ectodermal tissues must be present to account for all the changes observed in the syndrome.

S. R. GIFFORD.

AN UNUSUAL FAMILIAL SUPERFICIAL CHANGE IN THE CORNEA: REPORT OF A CASE. J. K. PAMEIJER, Klin. Monatsbl. f. Augenh. **95:** 516 (Oct.) 1935.

A boy aged 8 had a high degree of hypermetropia. The vision in his right eye, 2/60 of the normal, could not be improved with glasses, whereas the vision of 3/4 in the left eye was brought up to 5/4 by a convex lens of 8 diopters. The eyes were free from irritation, and the sensitiveness of the corneas was normal. The corneal microscope revealed that the superficial layers of the corneas were strewn with minute and fine gray spots and dots. Some of them were round; others had the shape of a pointed angle. The peripheral region of the cornea, near the limbus, showed only a few isolated dots. A narrow strip in the nasal and lower quadrants of each cornea was entirely clear in the boy and in four other members of the family. The form and size of these strips varied in the five patients. The dots appeared to be minute vacuoles or blisters with a double outline and either a clear center or a clear periphery. The substantia propria and the endothelium were normal.

The mother presented similar but larger vacuoles, some of which stained green with fluorescein; she complained of epiphora and photophobia for many years. The mother, her sister and two of the latter's four children had the peculiar clear strips. No connection existed between the clearly visible corneal nerves and the opacities. Two brothers of the mother were allegedly afflicted with the same malformation; they could not be reached for an examination. Green staining was obtained only in the two women, not in the children. Epithelial dystrophy (Fuch's) and nodular and lattice-shaped degeneration did not exist, nor were symptoms of avitaminosis present.

K. L. STOLL.

REMOVAL OF FOREIGN BODIES FROM POSTERIOR LAYERS OF THE CORNEA. G. G. BURSUK, Sovet. vestnik oftal. **7:** 154, 1935.

Bursuk constructed a knife for removal of amagnetic foreign bodies from the posterior layers of the cornea when routine measures are of no avail. The knife, when entered into the anterior chamber, adheres firmly to the posterior surface of the cornea and thus supports the foreign body and prevents it from slipping into the anterior chamber while it is being obtained from the external surface. It also prevents any possible injury of the iris or lens.

A description of the knife and of the technic of its use is given. It was tried out successfully in several cases in Bursuk's clinic. The slit lamp is essential for localization of the foreign body.

O. SITCHEVSKA.

Experimental Pathology

EXPERIMENTAL PTOSIS IN PRIMATES. W. MAHONEY and D. SHEEHAN, Arch. Neurol. & Psychiat. **35:** 99 (Jan.) 1936.

This study was prompted by an incidental observation in monkeys of a rapid recovery from the ptosis that follows experimental section of

the third nerve. The authors show that Müller's muscle, through sympathetic innervation, overcame the ptosis produced by intracranial section of the third nerve in from two to three weeks in monkeys and in a much shorter time in cats and dogs. After section of the nerve, excitement caused excessive widening of the palpebral fissure, presumably because of lack of inhibition from the third nerve.

After removal of sympathetic influence in addition to section of the third nerve, complete closure of the lid persisted until regeneration of some fibers of the third nerve had occurred. This regeneration was demonstrated by anatomic studies and was found to be nearly complete in three months.

After section of both the third and the sympathetic nerve it was observed that extreme fright or injections of epinephrine caused widening of the palpebral fissure, greater on the side of the operation than on the intact side. This paradoxical response is compared by the authors to the paradoxical dilatation of the pupil after section of the sympathetic nerves reported by Byrne and others. It is presumably the result of the action of epinephrine on a neuromuscular mechanism sensitized through being deprived of its sympathetic nerve supply.

Section of the seventh nerve and observation of paradoxical responses after degeneration of the sympathetic nerves over a longer period than is reported here would be of additional interest. However, the presentation of the work that has been done is pleasingly concise and scientifically instructive.

R. IRVINE.

General

OPHTHALMOLOGY IN THE WORLD WAR. A. JESS, München. med. Wchnschr. **82:** 1941 (Dec. 6) 1935.

During the war of 1870-1871, only 0.86 per cent of the injuries were injuries of the eyes; in the Chinese-Japanese war of 1894, only 1.18 per cent; in the Russo-Japanese war of 1904, only 2.22 per cent, while in the World War the figure reached 10 per cent. It was due to this fact that field stations for the treatment of ocular injuries were established, and many new and unusual procedures in the management of injured eyes were of necessity carried out by ophthalmologists. Fear of sympathetic ophthalmia caused them to enucleate many eyes. The necessity of having a strong magnet in the stations for removal of metallic foreign bodies from the injured globes was emphasized. Hemorrhage into the globe, luxation of the lens, rupture of the iris and choroid, detachment of the retina and other obscure injuries due to force were often entirely overlooked because of the general condition of the soldiers. Injuries to the brain in some cases caused blindness by interference with the centers of vision. The cornea and conjunctiva were often affected by gaseous fumes. Burns from explosive shells occurred frequently. Methyl alcohol poisoning was seen with its primary attack on the papulomacular bundle. Many chemicals were used by the soldiers in attempts to cause injury to their own eyes and thus escape service. Alum, soap, tobacco, paprika, chalk and other substances were tried. Night blindness due to avitaminosis was of frequent occurrence. Several cases are cited. Trachoma in its various phases was present and was

often adduced to gain exemption from service. Refraction and correction of the numerous near-sighted persons in the German army frequently called for the ingenuity of the oculist. Color blindness prevented proper recognition of color signals. Additional diseases encountered were all the more or less harmless ophthalmic conditions seen in every day life. In Germany 3,200 were blinded during the period of the war.

L. L. MAYER.

General Diseases

THE FUNDUS OCULI IN DIABETES MELLITUS. M. L. FOLK and S. SOSKIN, Am. J. Ophth. **18:** 432 (May) 1935.

Folk and Soskin give the following summary and conclusions:

"1. A study of the fundi of 150 diabetic patients and of a control group of 150 subjects without diabetes is reported.

"2. The correlation of certain retinal conditions, with the age, duration, and severity of diabetes and diabetic complications is brought out.

"3. The incidence of retinitis in those affected with diabetes is greater than is commonly believed.

"4. Diabetic retinitis is a definite entity and occurs in diabetes in the apparent absence of its complications.

"5. The incidence of sclerosis of the retinal vessels is but little higher in persons with diabetes than in those without in the same age groups, and when present is usually less severe.

"6. The prognosis of diabetic retinitis is comparatively good, provided the patient is properly managed and cooperates well.

"7. Effective diabetic therapy should lessen the incidence of retinitis.

"8. Frequent fundus examinations are of particular value to the diabetic patient."

W. S. REESE.

POSTGRIPPAL DISEASES OF THE EYE. S. M. KHORDAS, Sovet. vestnik oftal. **7:** 305, 1935.

Khordas selected a group of thirty-one patients whose eyes were affected during a severe epidemic of influenza in the years 1930 and 1931. Most of the patients were from 20 to 30 years old; the male sex prevailed. The cornea was most frequently affected, usually in the period from the seventh to the eleventh day after the grip. The clinical picture characteristic of herpetic keratitis was usually observed: photophobia, diminution of corneal sensitivity, ciliary injection and the presence of a number of small vesicles along the limbus. There were also recorded two cases of iritis, one of iridocyclitis, two of tenonitis, two of scleritis and three of unilateral paralysis of the abducens nerve.

Khordas believes that all postgrippal ocular complications are caused by the virus of herpes, that the virus of grip, by reducing the resistance of the body, activates the virus of herpes, and that thus complications of the eye set in.

Daily intravenous injections of a 40 per cent solution of methenamine (urotropin), because of the good therapeutic results from the use of

this drug in encephalitis, were tried in the treatment of these complications of the eye. The initial dose was 2 cc., and the amount was increased daily until a dose of 7 or 8 cc. was reached; the total number of injections varied from fifteen to twenty-five. The corneal disorders responded well to the treatment, as the sensitivity was restored and healing of the infiltrates took place after the first few injections.

O. SITCHEVSKA.

Glaucoma

EPINEPHRINE BITARTRATE: USES OTHER THAN IN THE TREATMENT OF GLAUCOMA. F. C. CORDES and D. O. HARRINGTON, Am. J. Ophth. **18:** 451 (May) 1935.

Cordes and Harrington draw the following conclusions: "Epinephrine bitartrate is a relatively little-used drug which deserves a prominent place in the ophthalmologic pharmacopeia.

"In addition to its action of lowering tension in chronic simple glaucoma, it has been found useful as a mydriatic for:

"1. Examination of the fundi in eyes with high normal tension.

"2. Examination of the fundi in glaucomatous eyes which are under the influence of a miotic.

"3. Powerfully dilating the pupil with the separation of posterior synechia in cases of old or neglected iritis.

"4. Dilating the pupil during the operation for congenital, soft, membranous or traumatic cataract.

"The marked vasoconstrictive action of this drug, which exceeds that of adrenalin, makes it an ideal hemostatic in the removal of chalazia, in cyclodialysis, and in the operation for retinal detachment, in which a dry field is essential to success."

W. S. REESE.

A CRITIQUE OF GLAUCOMA OPERATIONS. H. S. GRADLE, Am. J. Ophth. **18:** 730 (Aug.) 1935.

This paper does not lend itself to abstracting. Gradle considers operations for glaucoma in three divisions: (1) those to restore normal paths of drainage, (2) those to open new paths of drainage and (3) those to form paths of extra-ocular drainage. He concludes with a description of a new procedure to be used in the treatment of absolute glaucoma, namely, diathermic coagulation of the vitreous. He has used this in six cases, and in none was there recurrence of pain although the reduction of tension was only temporary.

W. S. REESE.

LATE INFECTION FOLLOWING A FISTULIZATION OPERATION FOR GLAUCOMA. D. CATTANEO, Ann. di ottal. e clin. ocul. **63:** 481 (July) 1935.

After a fairly complete review of the literature, Cattaneo describes a case in which infection developed eighteen years after a trephining for secondary glaucoma. The anterior chamber became filled with pus, and a ring abscess of the cornea developed rapidly. Pneumococci were found in smears and cultures of aspirated aqueous. Sections of the enucleated

eye showed the same organisms in the vitreous, and they were also present in the conjunctival secretion. Sections showed the route of infection from the conjunctivä over the trephine opening to the corneal stroma and intra-ocular contents.

S. R. GIFFORD.

THE RISE IN INTRA-OCULAR PRESSURE IN ACUTE ANGIONEUROSIS OF THE CILIARY BODY (GLAUCOMA ALLERGICUM) AND ITS RELATION TO CYCLITIC AND HETEROCHROMIC GLAUCOMA. ERNST KRAUPA, Arch. f. Augenh. **109:** 416, 1935.

By serous cyclitis one refers to a condition characterized by many opacities of the vitreous and deposits on the posterior surface of the cornea without the formation of posterior synechiae or visible nodules in the iris. It is frequently associated with a rise in intra-ocular pressure (secondary glaucoma). Frequently after the pressure has been reduced one may find central or peripheral areas of choroiditis. The disease is nearly always of tuberculous origin. With successful management of the secondary glaucoma the disease can nearly always be cured by foreign protein therapy and by the use of the roentgen rays. After the condition has been present for some time the iris as a rule becomes discolored so that it is difficult to differentiate the condition from the so-called Fuchs type of heterochromia.

In the latter condition miotics are also not indicated, as the glaucoma disappears if the anterior chamber is opened. Although Kraupa admits that the cause of Fuchs' heterochromia is not known, he considers in a rather vague way that it is due to some constitutional disturbance in the metabolism of the ciliary body. He likens this disease to constitutional albuminuria. A general discussion of heterochromia is given, with particular emphasis on Passow's views. The latter considers Horner's syndrome as a motor disturbance, heterochromia as a trophic disturbance and Fuchs' heterochromia as a vasomotor disturbance in the sympathetic nervous system, and likens these to masked forms of syringomyelia.

Four cases of glaucoma and cyclitis are reported, and a rather confused general discussion follows as to the possible relation between these two pictures. Kraupa looks on these cases as representative of a group in which, through some vasoneurotic disturbance of the ciliary body, protein is poured into the aqueous, with resulting increase in pressure. He prefers to use the term "glaucoma allergicum" for this condition.

F. H. ADLER.

Hygiene, Sociology, Education and History

ILLUMINATION INTENSITIES FOR READING. M. A. TINKER, Am. J. Ophth. **18:** 1036 (Nov.) 1935.

Tinker reviews the work done on intensities of illumination for reading. He concludes that in the light of experimental data the following specifications for intensities of light should fulfil the requirements of hygienic vision for the reading of legible print by the normal eye: 3 to 5 foot-candles with direct lighting and poor diffusion; 5 to 10 foot-

candles with the combinations of direct and semi-indirect illumination frequently found in homes and offices; 10 to 15 foot-candles with the better degrees of diffusion found in a few homes and offices. For defective eyes and the discrimination of fine details the intensities should be much higher.

W. S. REESE.

Lacrimal Apparatus

STRICTUROTOMY AND PROBING IN THE TREATMENT OF LACRIMAL DISEASE. R. SÁENZ-ALONSO, Arch. de oftal. hispano-am. **35:** 449 (Sept.) 1935.

VALUE OF CONSERVATIVE TREATMENT IN LACRIMAL DISEASES. MÁRQUEZ, Arch. de oftal. hispano-am. **35:** 455 (Sept.) 1935.

These two papers, published together, plead against the systematic discard of this method of treatment in appropriate cases, Sáenz-Alonso proposing several modifications in the procedure recommended by Poulard and Morax.

C. E. FINLAY.

TREATMENT OF EPIPHORA WITH ALCOHOL INJECTED INTO THE LACRIMAL GLAND. P. E. TIKHOMIROV, Sovet. vestnik oftal. **7:** 452, 1935.

The treatment was applied in stenosis of the duct or in hyperfunction of the lacrimal gland in which routine measures were of no avail. From 0.3 to 0.7 cc. of an 80 per cent solution of alcohol was injected into the palpebral part of the lacrimal gland. The average number of the injections varied from one to three. Edema of the lids and conjunctiva was observed for from three to five days. Ptosis usually persisted for from ten to fifteen days. Some patients complained of anesthesia of the temple and lids for a few days. Seventy-two patients (80 eyes) were treated with injections of alcohol for a period of two years; the patients were observed for from one to twelve months. Favorable results were obtained in 51 per cent. In thirteen patients there was a complete recurrence of the epiphora.

Alcohol was injected into the lacrimal glands of three dogs. The glands of the experimental dogs and one human gland were extirpated thirty-five days after the injections and examined microscopically. None of the four glands showed atrophy of the tissue. Tikhomirov therefore thinks that the alcohol affects the secretory nerves and causes a decrease in the function of the lacrimal gland. This explains the positive results observed as soon as three days after the injections and the recurrence of the epiphora, which could not recur in an atrophied gland. He states the following conclusions: 1. The injection of alcohol into the lacrimal gland is harmless. 2. In the treatment of recurrent epiphora repeated injections can be given. 3. Positive results were observed in 51 per cent of the cases. 4. Further observations are necessary to judge whether these results are permanent.

O. SITCHEVSKA.

Lens

TREMULOUS LENS. A. W. D'OMBRAIN, Brit. J. Ophth. **20:** 22 (Jan.) 1936.

Iridocyclitis developed in a man 50 years of age with an almost mature cataract. With the slit lamp the lens was found to be tremulous. The iris was not tremulous, so there was no dislocation of the lens. On the slightest movement of the eye the lens trembled as if it were suspended on an elastic string. The phenomenon is attributed to fluidity of the vitreous.

The author could find no record of a similar case in the literature.

W. ZENTMAYER.

COMMENTS ON ONE HUNDRED AND FIFTY ATTEMPTS AT SYSTEMATIC INTRACAPSULAR EXTRACTION OF CATARACT. H. LAGRANGE, Ann. d'ocul. **172:** 1003 (Dec.) 1935.

Lagrange, in 1930, in *Le bulletin médical,* published an article relative to an inquiry as to the best procedure for the extraction of cataracts. He now reviews the present technic, and, as in his published article on sclerotomy and on the operative treatment of strabismus, his comments have as their objective a research on simple technic. He states that it was the Arruga forceps that enabled him to proceed with this investigation.

He pictures (1) the circumstances that lead to faults in technic and (2) the operative results, and here dwells particularly on the postoperative complications. The faults to which he has alluded consist in an inability to grasp the capsule, the total extraction of a broken capsular sac not accompanied by its contents and the appearance of the vitreous body. The operative results are tabulated—first those in a series of sixty-one cases of intracapsular extraction combined with iridectomy in which the keratotomy was done with the Graefe knife. In a second series of forty-two cases, the keratotomy was done by puncture. In the third series of forty-seven cases, there was no iridectomy, and the keratotomy was performed by puncture. The visual results and other details are given for each of these series. The article has nine excellent illustrations.

S. H. MCKEE.

PERFECTED CORNEAL SUTURE FOR EXTRACTION OF CATARACT. S. BALDINO, Rassegna ital. d'ottal. **4:** 538 (July-Aug.) 1935.

Kalt's and Liégard's methods of suture, carried out before corneal section, are made unsatisfactory by a resulting depression of the edges of the wound.

Williams' and Czermak's methods of suture, employed after corneal section, fail to get perfect apposition of the edges of the wound.

Baldino passes his needle through the corneosclera before corneal section. The needle is not drawn through; it remains in situ. The Graefe section is then made as usual, but only up to the needle. The latter and its thread are then pulled through. With a sharp hook the corneal section at the upper pole is completed, and the thread between

the edges of the wound is seized by a blunt hook and pulled out in a loop large enough to allow extraction of the cataract. It is then tied, thus securing perfect anatomic apposition. V. R. SYRACUSE.

Lids

OPERATION FOR ENTROPION AFTER VOGT'S METHOD. H. SCHLÄPFER, Klin. Monatsbl. f. Augenh. **94:** 610 (May) 1935.

Vogt practiced for many years, but never before published a report on, a simple operation for spastic entropion. It is based on the principle of replacing the margin of the lower tarsus, turned faultily toward the eyeball, in its normal position. This is done by pushing the margin of the upper tarsus off the eyeball by pulling the temporal extremity of the lower lid in front of the upper lid. Thus the margin of the upper lid is placed between the margin of the lower lid and the globe at the temporal canthus. The older methods were devised with the aim of bringing the margin of the lower lid toward the globe so that the entire inner surface of the tarsus was in close contact with the globe after the operation. A liberal canthotomy is done down to the bone at the temporal canthus after the lids are pulled apart. Then a suture is laid through the external portion of the upper lid and out through the conjunctiva and the bulbar portion of the lower lid so that the suture appears in the bulbar portion of the canthotomy wound of the lower lid. After the suture is tied the wound surface of the upper lid must touch the conjunctival surface of the lower lid, the ventral margin of the wound of the upper lid must touch the dorsal margin of the wound of the lower lid, and the former canthus of the upper lid is placed behind the lower lid. This method is recommended for extensive entropion and those cases in which previous operations have failed.

K. L. STOLL.

Methods of Examination

NEWER DEVELOPMENTS IN PHOTOGRAPHY OF THE EYE. W. A. MANN, Am. J. Ophth. **18:** 1039 (Nov.) 1935.

Mann discusses external photography and photography of the fundus. Under the former he takes up black and white representation, color and infra-red photography, and under the latter, moving pictures and color work. He favors the Finlay color method and enumerates its advantages. Infra-red photography of the fundus has not been satisfactory.

W. S. REESE.

SPECTRUM ANALYSIS AS A RELIABLE METHOD IN PROVING THE PRESENCE OF METAL IN AND AROUND THE EYEBALL: REPORT OF A CASE. ROLF SCHMIDT, Klin. Monatsbl. f. Augenh. **95:** 440 (Oct.) 1935.

Only a few cases have been reported in which the presence of metal foreign bodies in the eyeball and in its neighborhood was proved by means of spectrum analysis. Schmidt adds the case of a man who suffered an injury to his left eye during the war in 1918. Amaurosis fol-

lowed, and chronic inflammation made the removal of the eye necessary in 1925. The patient complained of headache and of pain in the orbit for about two years, during which a 1 per cent solution of silver nitrate was used by another oculist. This treatment was discontinued when a slight brownish discoloration developed in a limited area of the conjunctiva. The oculist accused the patient of secretly using some coloring substance when the size of the brown discoloration increased. It had a diameter of 9 mm. when Schmidt admitted the patient to the University Eye Clinic in Freiburg. No pathologic signs could be observed in the conjunctival sac aside from slight congestion. The roentgenogram, however, revealed two foreign bodies, each the size of a pinhead; one was located near the base of the zygomatic process; the other, in the nasal portion of the roof of the orbit. A piece of the discolored conjunctiva was excised; it contained no melanosis or silver. Spectrum analysis of a small portion of conjunctival tissue proved strongly the presence of the silver lines 3281 and 3383 A and less distinctly that of the copper lines 3247 and 3274 A; furthermore, it revealed traces of calcium, probably remnants of dust, and traces of iron, which may have been the residue of blood. Argyrosis was therefore present, although the other tests had failed to discover it. The presence of the copper lines was explained by the common admixture of copper in silver nitrate pencils. The patient was convicted of having used a silver nitrate pencil to obtain a higher compensation as a war veteran.

K. L. STOLL.

Neurology

ENCEPHALOTRIGEMINAL ANGIOMATOSIS. M. APPELMANS, Arch. d'opht. **52:** 835 (Dec.) 1935.

That there is a distribution of cutaneous angiomas along nerve supplies in some instances has been known for a long time, but the association of "trigeminal nevi" with intracranial lesions was first pointed out by Kalischer in 1897. These nevi are often accompanied by glaucoma. The detailed description of a personal observation is presented, with photographs. The origin of nevi is discussed from an embryologic point of view and in relation to the sympathetic innervation. A table of twenty-four cases demonstrates the frequency of homolateral glaucoma and other ocular complications. According to van der Hoeve, Lindau's, Bourneville's and Recklinghausen's diseases belong in the same group. The differentiating factor is the embryologic stage at which they start. In conclusion, Appelmans believes (1) that angiomas of the area of the skin supplied by the upper two branches of the trigeminus nerve are very often associated with glaucoma and intracranial angioma; (2) that ocular and intracranial lesions co-exist frequently with nevi of the face which present no clearcut disposition in relation to nerve roots; (3) that the term "trigeminal angioma" of the face is inadequate, and "neurocutaneous angiomatosis" is more exact; (4) that roentgenograms of the skull should be made in all cases of extensive angiomatosis of the face, and (5) that the glaucoma associated with angiomatosis is of neurovascular origin.

S. B. MARLOW.

A New Proof of Perimetric Polymorphism in Opticochiasmal Arachnoiditis. J. Malbrán, Arch. de oftal. de Buenos Aires **10:** 634 (Aug.) 1935.

This is the report of a case in which a limitation of the visual field of the right eye to the upper nasal quadrant (with good central vision) and an irregular defect in the horizontal meridian over the temporal portion of the macular region in the left eye led the author to diagnose erroneously a meningioma of the tuberculum sellae ossis sphenoidalis. An operation revealed an opticochiasmal arachnoiditis, more pronounced around the right optic nerve. Death occurred in coma forty-eight hours after the operation. No intracranial explanation was found at autopsy.

C. E. Finlay.

Orbit, Eyeball and Accessory Sinuses

Physiological Considerations in the Treatment of Pulsating Exophthalmos. G. M. Dorrance and P. E. Loudenslager, Am. J. Ophth. **17:** 1099 (Dec.) 1934.

Dorrance and Loudenslager have studied the original reports of cases recorded since 1908 and comment on these. They discuss blood currents and blood vessel changes associated with arteriovenous fistula. They remark the reversal of current in the external carotid after ligation of the common carotid as not being generally known. They decry preoperative compression of the carotid vessels and simultaneous ligation of the internal jugular vein. In the presence of infection of the mouth, nose or throat the cervical wound should be drained. Cerebral accidents following ligations are largely due to sudden extreme reductions in blood pressures, resulting in stagnation of the blood in cerebral areas. They reach the following conclusions: "An arterio-venous communication between the internal carotid artery and the cavernous sinus should be expected to exhibit the same phenomena of hemodynamics observed in arterio-venous communications elsewhere in the body.

"On the basis of such expectation, ligation of the proximal artery—in this situation, the internal carotid—would produce a reflux of blood from the artery distal to the fistula, keeping active the fistula and possibly causing cerebral complications.

"Ligation of the common carotid, on the other hand, should be recognized as a partial ligation only of the internal. Because of the reversal of flow in the external, the internal becomes a branch of the external with about fifty per cent reduction in volume flow.

"Fractional ligation of the internal carotid in this manner presents a more satisfactory record chiefly from the standpoint of fewer cerebral sequelae. We submit that this may be accounted for by the probability that the partial reduction in blood pressure in the internal carotid is insufficient to start a backflow from the distal artery. And, further, if this backflow does not occur, the vessel will shrink in size, reducing its lumen and thereby reducing the size of any fistula present."

W. S. Reese.

BILATERAL ORBITAL TUMOR WITH EXOPHTHALMOS CURED BY MERCURIAL TREATMENTS. M. KALT, Arch. d'opht. **62:** 655 (Sept.) 1935.

An adult, 35 years old, previously well, acquired in the space of a month bilateral exophthalmos with complete immobility of the eyeballs and lagophthalmic keratitis due to a tumor involving both orbits. One series of mercurial treatments failed to check the progression of the exophthalmos on the right side. Bilateral biopsy showed a chronic inflammatory condition of the tissue, which was dense fibrous tissue with a perivascular infiltration of lymphocytes. On further intensive mercurial treatment the masses subsided. Inflammatory tumor of the orbit is not uncommon, but it is exceptional to see it bilateral and symmetrical. Kalt believes his case to be one of orbital syphiloma with osteoperiosteal localization and sclerogummatous invasion of the orbit, the point of origin being in the lacrimal glands. The Wassermann tests were repeatedly negative. The effect of antisyphilitic treatment was delayed. Biopsy was extremely important in the management of the case.

S. B. MARLOW.

PULSATING AND INTERMITTENT EXOPHTHALMOS AND ITS NONSURGICAL TREATMENT. M. DE-PETRI, Riv. oto-neuro-oftal. **12:** 306 (March-April) 1935.

The diagnosis of exophthalmos due to orbital varices is not difficult in the typical case. If the condition is due to a cavernous aneurysm of the carotid artery, the diagnosis is more difficult, especially if there is no bruit. This may lead to improper therapy.

Therefore, instead of having first recourse to surgical treatment, De-Petri makes a deep intra-orbital and perivascular injection of 2 cc. of a 10 per cent solution of quinine and urethane in the direction of the superior ophthalmic vein. One must be sure that one does not have the needle in a vessel, that the technic indicated is closely followed and that one is familiar with the possible complications and with the results that should follow.

This method obviates the dangers and complications of surgical intervention; it is within reach of every specialist and is devoid of danger if properly carried out. It may be applied in many cases. In case of failure, surgical intervention is always in order.

V. R. SYRACUSE.

Physiology

NEW THEORY OF VISION OF RELIEF: THE PRIORITY OF PERIPHERAL POINTS OF THE RÈTINA. ALAERTS, Arch. d'opht. **52:** 320 (May) 1935.

In a previous communication Alaerts attempted to demonstrate the importance of certain points in the retina in vision of relief. In the present report he attempts to show that the peripheral points are of greatest importance in this function and describes some simple experiments which support his theory. He describes a case in which a patient reported that he had noticed a spot in front of his eye, close to him. The

year before he had noticed a spot some distance away. Ophthalmoscopic examination revealed a fresh hemorrhage in the temporal half of the retina and a pigmented patch in the nasal half. This observation is used to support his theory that the nasal portion of the retina gives rise to the impression of distance and the temporal half to that of things close by.

The conclusions which he draws from the observations he has made are: 1. The essential factor in vision of relief is the peripheral part of the retina. The macula acts as a point of reference. 2. The nasal part of the retina gives rise to the impression of distance and the temporal to that of nearness. 3. There is always a comparison by superposition of two differently named portions of the retina. 4. The portion of the retina where the visual impression is most centrifugal gives rise to the sense of relief. This is the priority of peripheral points.

The phenomenon of pseudoscopic relief is also used in support of the theory. This paper should be read in the original.

S. B. MARLOW.

NONGLAUCOMATOUS COLORED RINGS OR CIRCLES. A. POLACK, Bull. Soc. d'opht. de Paris, July 1935, p. 396.

The patient is a distinguished astronomer and therefore should be able to describe the phenomenon very clearly. Druault has attributed the colored circles envisioned by persons without glaucoma to changes in the lens. The patient described the same distribution of colors and the same radial striations of the same short diameter as glaucomatous patients have noted, except that the visibility was not augmented by mydriasis. Also the disposition of the fishing hooks which are noted when a stenopaic slit is used were not eccentric from the pupil. These hoops which Druault calls "segments of a circle" and which are presented clearly when the subject looks at a point of light are at the periphery of the visual fields. In Polack's case these were noted in the center of the field at the luminous point. They were not seen when the stenopaic slit was deviated from the center line of the pupil. The patient, aged 75 years, had an increase of myopia from 4.5 to 6.5 diopters in one year's time. The visual acuity remained the same. The fundus was normal. In the vitreous were floaters and a striated membrane at the pupillary plane. No hypertension existed even after the use of atropine. The cornea, aqueous and lens were normal. Perhaps the changes in the vitreous account for the colored circles? The work of Rochon-Duvigneaud on the structure of the lens may aid in comprehending the mechanism.

L. L. MAYER.

THE DURATION OF AFTER-IMAGES. J. ESCHER-DESRIVIÈRES and R. JONNARD, Compt. rend. Soc. de biol. **120**: 681, 1935.

The observer, after being in obscurity for a half hour, viewed through a binocular photographic shutter for one-tenth second a square of transilluminated opal glass and by a stop-watch subjectively measured the duration of his after-image. Under definite conditions of excitation, the duration of the after-image is fairly constant for the same person

and increases with the length of adaptation till a certain maximum is reached, but varies considerably with different persons and with the illumination of the test object.

J. E. LEBENSOHN.

Refraction and Accommodation

CHANGES IN OCULAR REFRACTION IN DIABETES. C. CASTRIGNANI, Ann. di ottal. e clin. ocul. **63:** 869 (Nov.) 1935.

A girl of 16 years complained of a sudden loss of vision. Vision was reduced to 1/10, and myopia of 4 diopters was present. The patient was diabetic and had received some treatment previously but then had returned to a normal diet. A week later vision was 4/10, and the myopia was reduced to —3.00. Ten days later the vision was 12/10 in each eye without correction.

Castrignani reviews the various theories in explanation of such refractive changes in diabetes but believes that the problem has not been solved.

S. R. GIFFORD.

AN EPIDEMIC OF BILATERAL PARALYSIS OF ACCOMMODATION IN CHILDREN. A. FUCHS, Wien. klin. Wchnschr. **48:** 1547 (Dec. 13) 1935.

Within a month's time after Oct. 10, 1935, Fuchs saw four girls with bilateral paralysis of accommodation. These were girls from 8 to 16 years of age. They had no complaints other than some blurring of vision, and in each case both the history and the general physical examination were unproductive of a cause. In each case the accommodation was reduced from 13 to between 3 and 4 diopters. The near point for accommodation was between 23 and 32 cm. The rarity of this condition, revealed in the fact that only one case, seen by Genet in 1934, has been reported in the literature, is the reason for this report. Such cases are found following cases of diphtheria and influenza, but because of the transient nature of the paralysis and because of the fact that oculists are not called to see the patients the cases are not often reported in the literature. Usually there is an accompanying paresis of one or more extra-ocular muscles as noted following encephalitis. The girls were not bacteria carriers, were in various parts of the country during the preceding summer and gave no history of coryza or other disease. The prognosis is at present unknown. In the postdiphtheritic type symptoms are absent after one week. A table considering the age, refraction, punctum proximum, accommodation and remarks accompanies the article.

L. L. MAYER.

Retina and Optic Nerve

THE OBLITERATION OF RETINAL TEARS BY DIATHERMOCOAGULATION. P. VEIL and M. A. DOLLFUS, Arch. d'opht. **52:** 162 (March) 1935.

The results of one hundred operations by diathermocoagulation, the earliest dating from July 1933, are reported. In one series the method of Weve or of Šafář was used; in a second, the pyrometric technic of

L. Coppez, slightly modified. The operations were done in a dark room under ophthalmoscopic control, for which a contact glass is not indispensable. The dose of current was regulated so as to obtain the minimum reaction visible to the ophthalmoscope. Perforating diathermocoagulation is easy to perform over a limited area. It is difficult if carried out over an extensive area. In the latter case surface coagulation is preferable. Veil and Dollfus prefer diathermic puncture for draining off the subretinal fluid rather than use of a knife. They have obtained cures in 55 per cent of cases by perforating diathermy and in 58 per cent of cases by surface coagulation with the pyrometric electrode. Cicatrization is much slower after electrocoagulation than after thermocautery or galvanocautery. Therefore the period of rest in bed should be much longer, and stenopaic glasses should be worn for at least one month.

S. B. MARLOW.

RETINAL ARTERIAL PRESSURE IN RELATION TO THE GENERAL CIRCULATION AND TO CERTAIN OCULAR DISEASES. E. MARCHESINI, Ann. di ottal. e clin. ocul. **63:** 532 (July); 621 (Aug.) 1935.

Records were made with the Bailliart dynamometer for a series of normal and a series of diseased eyes before and after administration of acetylcholine. In twenty-one normal eyes the average systolic pressure in the retinal arteries was 73.6 mm. of mercury, and the average diastolic pressure was 34 mm. The relation of these figures to those for the general blood pressure was approximately that found by Bailliart, the retinal systolic pressure being 54.23 per cent of the general systolic pressure and the retinal diastolic pressure 41.4 per cent of the general diastolic pressure. After administration of acetylcholine a drop in pressure occurred, averaging 6.42 and 4.04 mm. for the general systolic and the general diastolic pressure, while the retinal pressures dropped 3.42 and 2.19 mm. A slight increase in vision accompanied this drop in pressure, amounting to from .33/10 to .4/10. Patients with high myopia and those with hyperopia showed no differences in response as compared with normal persons. Patients with general hypertension, including one with thrombosis of a retinal artery, showed no appreciable drop in pressure after the administration of acetylcholine and no increase in vision. In patients with optic atrophy a normal drop in pressure followed the administration of acetylcholine, but there was no improvement in vision. In three patients with retinitis pigmentosa hypotony was observed in the retinal arterial pressure as compared with the general arterial pressure. The fall of pressure obtained with acetylcholine was almost twice the normal average, and vision was increased slightly. In six patients with retinal angiospasm relative hypotony was also observed, and the fall in pressure after the administration of acetylcholine was much greater than normal. The most marked increase in vision following the use of acetylcholine was observed in this group, the average amounting to 2.16/10 for right eyes and 1.5/10 for left eyes. Five patients with retrobulbar neuritis showed slight relative hypotony, a fall of pressure after the administration of acetylcholine which was about normal and marked increase in vision, though not so marked as in the previous group.

S. R. GIFFORD.

OCCLUSION OF THE CENTRAL RETINAL ARTERY. F. GABARDI, Rassegna ital. d'ottal. **4:** 333 (May-June) 1935.

Gabardi examines critically the possible causes concerned in occlusion of the central retinal artery (embolism, endarteritis obliterans, thrombo-arteritis, angiospasm) and the differential diagnosis. He presents five cases of partial or total occlusion.

A clinical differentiation of the causes cannot be easily outlined or predicated from clearly pathognomic signs. A differentiation is possible but only on the basis of a personal evaluation, and on this account it cannot be absolute. This failing should be an incentive for new, more accurate observations which will carry the observer beyond the usual diagnosis of embolism.

V. R. SYRACUSE.

PERIPHLEBITIS AND ENDOVASCULITIS OF THE RETINAL VESSELS; SCLEROTIC, TUBERCULOUS AND SEPTIC DISEASES OF THE CHOROID: REPORT OF CASES. W. GILBERT, Klin. Monatsbl. f. Augenh. **94:** 335 (March) 1935.

Gilbert refers to Marchesani's research on juvenile angiopathy of the retina, on recurring hemorrhages in the vitreous and on obliterating thrombo-angiitis as described by Buerger. He points out that Marchesani limited the frequency of tuberculosis as a cause in these cases, many of which he attributed etiologically to general diseases of another nature. He reports two of three cases in which he found tuberculosis absent but syphilis present, yet evidently other general diseases as the etiologic factors. The first case was that of a woman, aged 48, who suffered sudden failure of vision in her left eye in November 1933. Only a small hemorrhage in the retina, surrounded by an opaque area in this membrane, could be noted in the fundus. Antisyphilitic treatment failed to improve the vision. Pallor of the disk was noticed a few weeks later, simultaneous with abdominal spasms. Swelling of the left leg and then thrombosis in the right leg appeared in the latter part of January 1934. Vascular obliteration caused by endarteritis, i. e., thrombo-angiitis as described by Buerger, with consequent gangrene of the lower extremities was considered the cause of death, which occurred on Jan. 30, 1934. The disease began with obstruction of the central retinal vessel of the left eye; spasms in the abdomen followed, and vascular obliterations in the lower extremities caused the patient's death.

In the second case, that of a man aged 28, one eye presented typical perivasculitis with involvement of the retinal vein and artery, encased veins and hemorrhages in the area of the inferior temporal branch of the retinal vein. Full vision was recovered after three months. Tuberculosis was absent, but the Wassermann reaction was strongly positive. Thrombosis in the lower part of the left leg developed later and lasted for three weeks.

Both patients complained of a sensation of cold in their feet, one of the symptoms mentioned by Buerger and Marchesani.

Gilbert doubts the correctness of Marchesani's standpoint in excluding tuberculosis in all cases of this type. He refers to tubercle bacilli which he found in the walls of the veins and in their close surroundings and to giant cells.

In the second part of his paper he discusses the differential diagnosis of diseases of the choroid caused by nephritis, arteriosclerosis, tuberculosis and septic conditions. These considerations are worthy of notice but too detailed to be enumerated in an abstract. Two items may be mentioned: The first is the case of a woman, aged 63, who had severe inflammation of the anterior part of the uvea in one eye. Very firm posterior synechiae caused secondary glaucoma. After iridectomy an area of sclerosis of the choroidal vessels about 3 disk diameters in circumference was noted in the macula. Choroidal foci were absent. Similar conditions existed in the other eye. The cause of this disease, in Gilbert's opinion, was sclerosis of isolated areas of the uveal vascular system; tuberculosis, syphilis, rheumatism and infections could be ruled out.

As to disseminated choroiditis, he arrives at the conclusion that it is of tuberculous origin, and that its very presence is indicative of the tuberculous primary complex which gave rise to it.

K. L. STOLL.

Trachoma

PRELIMINARY NOTE ON THE PRESENCE OF INFRAMICROBIAL ELEMENTS IN TRACHOMA FOLLICLES. A. CUÉNOD, Arch. d'opht. **52**: 145 (March) 1935.

In this report the attempt is made to establish firmly the existence of intrafollicular bodies in trachoma. With few exceptions, due probably to faulty technic at the beginning of the study, these bodies have always been found in cases of active trachoma, several hundred in number. They are not only constant but exceedingly numerous and appear in two general forms—one extremely small, which corresponds to what has been called the "elementary corpuscle" in inclusions proper, and the other much larger, analogous to what has been termed the initial body. The technic employed in the search for these bodies in the trachoma follicle is described. In the absence of all other pathologic bacteria these bodies have so much in their favor that one must be careful not to attribute the disease to them. In a later work Cuénod hopes to present reasons for believing them to have an active, if not an exclusive, part in the causation of the disease.

S. B. MARLOW.

SUCCESSFUL TRANSMISSION OF TRACHOMA IN MAN. E. DELANOË, Rev. internat. du trachome **12**: 242 (Oct.) 1935.

The right eye of an Arab, blind since childhood, from variola, had since then occasioned no discomfort and was absolutely without signs of trachoma. His wife, however, was being treated for trachoma in a hyperacute form. A fine applicator covered with sterile cotton was well rubbed over her tarsal conjunctiva and then well rubbed on the tarsal conjunctiva of his blind eye. After a week itching, tingling and discharge affected the blind eye. Five weeks later, examination disclosed a succulent tarsal conjunctiva studded with follicles.

In two other blind eyes inoculation failed, probably because the secretion was merely applied, not thoroughly rubbed in.

J. E. LEBENSOHN.

INOCULATION OF TRACHOMATOUS MATERIAL INTO THE BRAINS OF GUINEA-PIGS. A. VON SZILY, Klin. Monatsbl. f. Augenh. **95:** 433 (Oct.) 1935.

Referring to previous experiments in intracranial transmission of herpes, von Szily reports on a series of similar experiments with trachomatous material. Guinea-pigs were used. Under slight ether anesthesia and after shaving and disinfecting a small area of the galea of a guinea-pig the skull was prepared free; a small opening from 1 to 2 mm. in diameter was drilled through the skull, and 0.1 cc. of a weak solution of trachomatous conjunctival tissue was injected into the brain. The wound in the galea was closed with sutures. No cerebral or general physical symptoms resulted. From two to three months later the skull was opened in a manner described in detail, and the brain removed. The site of the injection was barely visible, but numerous small gray nodules, ranging in size up to that of a millet seed, were noted in the meninges, especially in the median line of the convexity of the brain. Practically no reaction in the brains was observed histologically, and perivascular accumulations of cells such as those found in encephalitis were absent. A number of photomicrographs illustrate the histologic observations. Large isolated follicles were found in the meninges but separated from the blood vessels; they occurred in the dura mater and in the arachnoid and between these membranes. The results corresponded with those obtained by intra-ocular injections, and numerous germ centers in the shape of trachoma follicles were in evidence. The nervous elements were free from follicles, as they were absent in the retina after intra-ocular injections. The distribution of the follicles over a large area is indicative of the activity of the specific noxa.

K. L. STOLL.

Tumors

A CYLINDROMATOUS BASILOMA OF THE LOWER LID. G. W. CAOCCI, Ann. di ottal. e clin. ocul. **63:** 519 (July) 1935.

Caocci describes a basal cell carcinoma involving the lower lid, at the outer angle. The unusual feature was the presence of numerous cavities lined with epithelium. The cavities contained a substance with the staining properties of mucin. The growth apparently originated in a previously ectropionized conjunctiva, and the excessive formation of mucin was apparently responsible for the formation of cavities.

Photomicrographs and a bibliography accompany the article.

S. R. GIFFORD.

TUMOR OF THE CHOROID. M. LAIGNIER, Bull. Soc. d'opht. de Paris, June 1935, p. 357.

A man 35 years old consulted Laignier because of a small brown freckle on the iris of his right eye; the freckle appeared to be enlarging. The visual acuity was normal. Normal findings were recorded from the general examination. A large iridectomy including the mass was accomplished. The pathologic diagnosis was melanotic sarcoma. Four or five months later, when the fundus of the affected eye was examined,

there was noted at 6 o'clock, two disks' width from the ora serrata, a small white spot resembling an exudate. Roentgen examination and use of a magnet showed no foreign body in the eye. Transillumination indicated a large tumor. Subsequently the retina over the affected area became detached. Photographs of the fundus are shown. The patient refused removal of the eye. Laignier asks whether diathermy should be applied.

L. L. MAYER.

HEMOGEMANGIOMA OF THE UPPER EYELID WITH EXTENSION TO THE LOWER LID, TEMPORAL REGION AND ORBIT. A. NATALE, Arch. de oftal. de Buenos Aires **10:** 587 (Aug.) 1935.

Natale reports a case of hemogemangioma. He gives this name to the tumors formerly known as fibro-angiomas, fibrohemangiomas and fibrous hemangiomas which were under the generic name of elephantiasis, on account of their embryonic origin from successive vascular spurts.

The patient was a man 30 years of age, without personal or hereditary antecedents of this condition. A horseshoe-shaped tumor involved the upper eyelid and part of the lower eyelid as well as the temporofrontal region. The palpebral fissure was narrowed and lower than that of the other eye. The eyeball was pushed downward and inward. The cornea was covered by a xerotic secretion; the bulbar conjunctiva was red, swollen and infiltrated above by the yellowish tumor. The consistence of the tumor was soft, elastic and non-nodular.

Natale describes the clinical characteristics of angiomas and hemangiomas, distinguishing two varieties: (1) simple or telangiectatic and (2) cavernous, the latter being circumscribed or diffuse. The present tumor belonged to this variety.

The eye was treated surgically in four sessions, which are described in detail.

The pathologic examination revealed a fibrillar interstitial connective tissue enclosing new-formed capillaries which were distributed in a disorderly manner, with some mast cells.

The literature on the subject is reviewed on the basis that tumors of this type were formerly known as elephantiasis, the present one being almost identical with that described by van Duysen in 1889.

C. E. FINLAY.

TWO CASES OF RECKLINGHAUSEN'S DISEASE. F. J. SORIANO and H. PÍCOLI, Arch. de oftal. de Buenos Aires **10:** 620 (Aug.) 1935.

Soriano and Pícoli report two cases of painless tumor of the eyelid. In one instance spots of pigment and papillomatous nevi were found on the skin of the back and face. In both cases histologic examination showed fibroblastic cellular elements in a nestlike arrangement, which in transverse sections had an appearance similar to that of nerve fibers. In each case the neurofibroma was located in the supraorbital branch of the superior division of the trigeminus nerve.

The reports are preceded by a detailed account of the symptoms of Recklinghausen's disease when these differ from the classic triad and of the different ocular lesions possible. The literature on the subject is extensively reviewed.

C. E. FINLAY.

Uvea

DIAGNOSIS AND SPECIFIC TREATMENT OF TUBERCULOUS CHOROIDITIS. A. SAMOJLOFF and A. TIHOMIROVA, Ann. d'ocul. **172:** 113 (Dec.) 1935.

The ophthalmoscopic diagnosis of tuberculous choroiditis presents great difficulty. The authors have seen many cases in which ophthalmoscopic examination gave no indication of the cause of the choroidal disease, and it was only by successful treatment with tuberculin that the tuberculous nature of the disease was shown. The difficulty is due to the fact that the tuberculous process in the choroid in many cases does not present any specific sign. On account of these difficulties, it is the opinion of many authors that tuberculous lesions of the choroid are rare. The first cases of tuberculous choroiditis to be recognized were those found in subjects with tuberculosis, and it was in these cases that the tuberculous origin of the condition was first admitted. Also in cases of tuberculous meningitis the specific origin of choroidal infection was generally recognized. It was only at a much later date that similar choroidal lesions in cases of chronic benign tuberculosis were admitted to be due to this cause.

Systematic ophthalmoscopic examination of the eyes of thirty-three persons with tuberculous choroiditis (fifty diseased eyes) showed a focal reaction of the choroid after subcutaneous injection of tuberculin into all in which tuberculosis was present.

The article is illustrated by two charts. S. H. McKEE.

Vitreous

THE CYTOLOGY OF THE DISEASED VITREOUS. M. LÓPEZ ENRÍQUEZ, Arch. de oftal. hispano-am. **35:** 197 (April) 1935.

This is a preliminary report on the microscopic demonstration of the migration of retinal pigment into the vitreous in pathologic cases, with predominance of Hortega cells and with variations depending on accidental and local conditions. The author deduces from different authors' observations of the cellular element of the cerebrospinal fluid that in pathologic cases the Hortega cells can migrate into this fluid.

C. E. FINLAY.

DEGENERATIVE PROCESSES IN THE VITREOUS OF THE HUMAN EYE AND THE ORIGIN OF TEARS IN THE RETINA. G. BAURMANN, Klin. Monatsbl. f. Augenh. **95:** 259 (Aug.) 1935.

Baurmann examined the vitreous body in situ in four enucleated eyes with an especial technic. The ages of the patients were 16, 31, 34 and 55 years, respectively. Four distinct types of degeneration of the vitreous had occurred, leading to a formation of vacuoles within the vitreous. These changes in the vitreous increased with age. The formation of vacuoles, filled with a fluid, began with a typical transformation of the normal stringy structure of the vitreous into a coarse network of granular cords. This tendency of the vitreous to degenerate rela-

tively early and the anatomic fact that the vitreous is closely connected with its surroundings only in the area of the orbiculus ciliaris and the most anteriorly located portion of the pars optica of the retina, in Baurmann's opinion, form the base for the formation of some of the retinal tears. When the liquefaction of the vitreous has reached an advanced stage the tears in the retina occur in this manner: Isolated pearls of vitreous which adhere to the anterior portion of the pars optica of the retina move contrary to the motion of the liquefied contents in the vitreous chamber when the eyeball moves; traction resulting at the insertion of the pars optica causes the tears in the retina. Applying Brückner's measurements of the velocity of ocular motions, Baurmann found that a pearl of vitreous of the bulk of 0.1 Gm. will exert a traction equivalent to 0.3 Gm.

K. L. STOLL.

Sympathetic Ophthalmia

STATISTICS ON THE OCCURRENCE OF SYMPATHETIC OPHTHALMIA. B. WALDMANN, Arch. f. Augenh. **109:** 441, 1935.

Waldmann has already called attention to his theory that sympathetic ophthalmia is of ectogenous origin and that the exciting cause is to be found in the nasal accessory sinuses. He supposes that it reaches the eye from there along the pia mater of the optic nerve or the ciliary nerves.

The present article is concerned with further proof based on statistics from a number of clinics scattered over the world. In answers to a questionnaire he finds that, of 193 cases of sympathetic ophthalmia observed in these clinics, 146 occurred between October and April and only 47 between May and September. He interprets this as further evidence that the cause of sympathetic ophthalmia is associated with infections of the upper respiratory tract, since in these months such infections are more common.

The frequent occurrence of neuritis with the beginning iridocyclitis proves that the optic nerve plays a significant rôle in the dissemination of the infectious agent.

F. H. ADLER.

Therapeutics

PRACTICAL TREATMENT OF OCULAR TUBERCULOSIS. GÓMEZ-MÁRQUEZ, Arch. de oftal. hispano-am. **35:** 177 (April) 1935.

Gómez-Márquez considers practically the whole community as being infected with tuberculosis, primarily in the first months of life, and as reacting in the presence of reinfections according to the degree of immunity and allergy developed.

In cases of ocular reinfection of the iris, for example, high immunity with high allergy produces acute iritis with complete recovery; high immunity with low allergy, mild iritis with complete recovery; low immunity with high allergy, acute iritis with subsequent tubercle formation in the iris; low immunity with low allergy, mild iritis with tubercle formation in the iris. It is therefore essential, especially in the treatment of ocular tuberculosis, to determine primarily what elements of the

clinical picture depend, respectively, on the immunity and the allergic factors, and then to attack the germ, first directly and second indirectly, and finally to modify the existing allergic condition. For the first, the author relies on chemotherapy, preferring the gold salts, especially sanocrysin. For the second he uses the old treatment with iodine, arsenic and iron and change of climatic environment—the seaside for torpid conditions and moderate altitudes for erethitic and congenital ones, with prudent aeration and sunlight. For the third group he recommends a combination of these two forms of treatment and the administration of tuberculin if the lesions point to hypersensibility.

He further divides tuberculous lesions into three groups: (1) destructive lesions produced by metabolic action of the germ, without congestive exudative phenomena of tubercle production, in which chemotherapy and hygiene are indicated; (2) congestive and exudative lesions with tubercle production, due to hypersensibility, in which tuberculin should be used; (3) mixed lesions, in the treatment of which both procedures should be combined.

C. E. FINLAY.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Feb. 17, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

METHYLTHIONINE CHLORIDE (METHYLENE BLUE) STAINING OF THE EYE. DR. ROBERT K. LAMBERT.

Three patients presented an interesting and unusual appearance of the ocular fundus. On ophthalmoscopic examination, the fundus appeared as though seen through a blue filter. The patients had been receiving methylthionine chloride U. S. P. (methylene blue) by mouth over a long period. The eye of one of the patients was examined after death, and it was found that the vitreous had taken a blue stain.

DISCUSSION

MR. E. B. BURCHELL: When the eyeball was opened, the vitreous showed a much deeper shade of blue than it does at present, and as the process of dehydration went along the blue faded. The eye had been fixed in Bouin's solution, which consists mostly of tri-nitrophenol, and it is a question whether the entrance of the tri-nitrophenol into the globe was not the cause of the loss of some of the blue, as after the eye had been sectioned and mounted microscopic examinations revealed no blue pigment.

DR. ROBERT K. LAMBERT: When methylthionine chloride is injected into the body, it is largely reduced to a colorless form, but in some tissues it remains blue. It seems possible that the region around the retina, having such a high oxidative rate, kept the blue form to a greater extent. My co-workers and I had called to our attention the case of a man who died from carbon monoxide poisoning and who had received methylthionine chloride intravenously. The man's brain was colorless. When it was sectioned, however, and the cut surfaces exposed to the air, these rapidly turned blue as the methylthionine chloride was oxidized. We have no explanation to offer as to why the myelin fibers of that particular specimen did not turn blue.

HOMOKERATOPLASTY. DR. JAMES G. MORRISSEY.

Complete homokeratoplasty was done on a young woman of 23 who had a painful leukoma that had developed after keratomalacia at the age of 2 years.

THE VALUE OF THE CONTACT GLASS IN THE THERAPY OF LAGOPHTHALMIC AND NEUROPARALYTIC KERATITIS. DR. DANIEL M. ROLETT.

The contact glass as a therapeutic agent was tried with success on two patients with corneal ulcers. In one patient a serpiginous ulcer healed within ten days after he began wearing the contact glass filled with a strong protein silver in a concentration of from 3 to 5 per cent.

The second patient had a typical ulcer of the cornea due to lagophthalmos and neuroparalytic keratitis in which the usual conservative treatment was of no avail. With the contact glass filled with physiologic solution of sodium chloride and worn for various periods during the day and night, the ulcer cleared entirely within several weeks.

PATHOLOGIC ASPECTS OF CHRONIC INFLAMMATORY CONDITIONS OF THE CORNEA. DR. BERNARD SAMUELS.

By way of preparing the audience for a better understanding of the paper that Dr. Jacob Jacobson was to read, Dr. Samuels presented a number of microscopic preparations, which were of two types:

1. Lesions that Dr. Jacobson declined to treat, quiescent old nonvascular corneal scars, some of which showed degenerative changes in the way of calcareous deposits. In declining to attempt to treat these scars Dr. Jacobson explained that the nonvascular fibrous elements of which they consisted were not amenable to absorption.

2. The opacities accepted for treatment. These consisted of vascularized subacute infiltrations lying between the lamellae of the cornea, as in interstitial keratitis of syphilitic or tuberculous origin, and of pannus, especially trachomatous pannus.

RESULTS OBTAINED WITH BENZYL CINNAMATE IN THE TREATMENT OF TRACHOMA AND CORNEAL OPACITIES. DR. JACOB JACOBSON, Paris, France.

This article will appear in full in a later issue of the ARCHIVES.

March 16, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

THE YOUNG OPERATION FOR PTOSIS. DR. WILLIS S. KNIGHTON.

The Young operation for congenital ptosis of the right upper lid was performed on a young boy whose left eye was normal. The right superior rectus muscle was normal, but the levator palpebrae was paralyzed. An adhesion was made between the superior rectus muscle and the tarsus, forming a symblepharon, according to the technic of George Young. The ultimate result was good cosmetically and functionally.

DISCUSSION

DR. W. GUERNSEY FREY: I should like to ask whether there is any interference with lateral movements of the eyeball after this operation.

DR. SIGMUND A. AGATSTON: After hearing Dr. Knighton describe the operation, I tried it on a patient two and a half weeks ago. The result so far looks very good.

DR. WILLIS S. KNIGHTON: In reply to Dr. Frey, as far as one can see, there is no interference with lateral movements of the eyeball. If one observes the patient carefully one may notice a little twitching of the eyelid when the eye turns right or left, but the patient does not complain of any interference.

BILATERAL VITREOUS CYST. DR. CHARLES A. PERERA.

Twenty instances of a unilateral cystic structure in the vitreous have been reported, but no one has had the opportunity of studying such material histologically. The origins of these bodies have been more or less speculative, although in eight of the twenty-four cases that have been recorded the cysts appeared to be definitely related to persisting congenital structures. The possible derivations of the cysts include: the fetal ocular cleft, mesodermal elements of the embryonic vitreous, the hyaloid apparatus, a cyst of the ciliary process, formed exudate in the vitreous, products of retinal degeneration or proliferation, and degeneration of a parasitic cyst.

ORBITAL TUMORS. DR. C. S. O'BRIEN.

This paper will be published in full, with the discussion, in a later issue of the ARCHIVES.

April 20, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

SIMPLE CONJUNCTIVAL SUTURE FOR USE IN SECTION FOR REMOVAL OF A CATARACT. DR. S. A. AGATSTON.

A suture to safeguard the eye in the course of operation should be simple and should be capable of being introduced with ease and speed and fastened with dispatch. A small V-shaped conjunctival flap is made, and the needles, 2 mm. apart, are passed through the flap and then through the conjunctiva above. The suture is not pulled tight, a lateral loop being left on each side, out of the way of the incision. Immediately after delivery of the lens, the assistant effects a closure in purse-string fashion by traction on the two parallel threads so that the flap slides under the conjunctiva above. Mere traction closes the wound safely, and the suture can be tied at the surgeon's leisure.

This method has been used during the past three years in all such operations on my private and public patients and by Dr. Bernard Samuels in his service at the New York Eye and Ear Infirmary.

DISCUSSION

DR. EUGENE BLAKE, New Haven, Conn.: It seems to me that failure to put the suture through the episcleral tissue sacrifices support and security.

DR. SIGMUND A. AGATSTON: I do not think it is necessary to pierce the episclera, because with the lower flap sliding under the upper the suture is just as strong as though the needle went into the episclera.

TWO TUMORS OF THE EYE: (1) AMPUTATION NEUROMA OF THE LONG POSTERIOR CILIARY NERVE. (2) PLEXIFORM NEUROMA OF THE CHOROID IN A NONBUPHTHALMIC EYE. DR. LOUISE H. MEEKER.

1. The amputation neuroma is in an eye that shows "phthisis bulbi": The patient, a 19 year old boy, has been blind since birth—an instrumental delivery. All the tunics of the eye were ruptured near the optic nerve. The nerve is enlarged with ganglions at several points. The long posterior ciliary nerve of the other side forms an intrascleral loop beneath the conjunctiva, indicating a congenital tendency toward growth. This tumor links the so-called neurofibroma of Axenfeld with that of Kyrieleis and duplicates amputation neuromas of other peripheral nerves. There are no associated evidences of Recklinghausen's neurofibromatosis.

2. The plexiform neuroma is in an eye removed for corneal ulcer and hypopyon with glaucoma. The patient, a woman, aged 41, gave a history of iridocyclitis of two years' duration which followed a blow on the eye. The eye is of normal size. The filtration angle is blocked by the adherent iris, and the disk is excavated. The choroid is thickened posterior to the equator, and the normal stroma is partly replaced by nerve fibers, which are greatly increased in number about the posterior ciliary vessels. Ganglion cells are very numerous, especially a few millimeters from the papilla. This finding in a nonbuphthalmic eye has been reported once (Freeman). The patient's other eye is apparently normal, and there are no further evidences of Recklinghausen's neurofibromatosis.

LESIONS OF THE OCULAR FUNDUS IN PATIENTS WITH ESSENTIAL HYPERTENSION AND ARTERIAL AND RENAL DISEASE. DR. MARTIN COHEN.

This paper may be presented in summary as follows:

1. The clinical symptoms of these patients are correlated with various lesions of the fundus.

2. The lesions of the fundus in some of the patients with essential hypertension simulated those seen in the patients with nephritis, arterial sclerosis, toxemia of pregnancy and diabetes.

3. In the patients with malignant essential hypertension, however, the lesions of the fundus were more severe than those found in the patients with the other diseases considered, with the exception of those with nephritis of certain types. These lesions consisted mainly of marked venous stasis, arterial contraction, profuse hemorrhages, throm-

boses, white plaques, macular changes and edema of the optic disk. These findings were accompanied by normal or practically normal renal function, differentiating malignant essential hypertension from nephritis in a clinical sense.

4. Hypertension was noted as a common feature in all of these patients; accordingly, it was difficult, if not impossible, to state from the observations in the fundus which was primary.

5. Edema of the optic disk in association with malignant essential hypertension is significant of its end-stage, which terminates in nephritis, causing a grave prognosis regarding the life of the patient.

6. It is difficult to decide from the lesions of the fundus in the patients with essential hypertension when the malignant phase first appeared and what its existing stage is.

7. There may be a similarity between the picture of the fundus in the terminal stage of malignant essential hypertension and that observed in chronic glomerulonephritis.

8. Further microscopic studies of the retinal and choroidal vessels are necessary in order to keep abreast with the more recent investigations of the arterioles of the kidneys and other organs.

9. The differing opinions among clinicians regarding the concept of essential hypertension cause various interpretations of the pictures of the fundus. A uniform clinical concept would establish more firmly the correlation of these pictures with the observations made by internists and pathologists.

10. The number of cases presented is too small to allow one to draw definite conclusions, and the report is presented merely to stimulate further studies in this important field.

DISCUSSION

DR. HERMAN O. MOSENTHAL: Those interested in internal medicine are just beginning to appreciate the vast significance of the pathology of the blood vessels. About twenty years ago, when arteriosclerosis, thrombo-angiitis obliterans, Raynaud's disease and periarteritis nodosa were regarded as almost the only diseases of the blood vessels, the situation was static because the vascular tree was looked on largely as a system of inert tubing rather than one composed of vital tissue with a special function, subject to somewhat the same diseases that occur in the lungs, the kidneys, the gastro-intestinal tract and elsewhere. Many vascular diseases have been found since that time and are still being studied, and when Dr. Cohen says he wishes that the clinician would give him a well conceived uniform description of what the vascular changes in hypertension are, he can only be furnished with an inconclusive partial statement. It was less than twenty-five years ago that Sir Clifford Allbutt in England and Theodore Janeway in America distinguished between senile, i. e., decrescent, arteriosclerosis and hypertensive arterial degeneration; since then the necrotizing lesions of the arteries, first discovered in the renal vessels by Fahr, have been recognized as something apart. All these arterial changes occur commonly in patients with hypertension, and it is easy to see how difficult it may be to get a uniform picture of the retina since it is dependent on the

many and variable vascular changes accompanying permanently increased arterial pressure.

The so-called benign and the malignant hypertension deserve mention because these terms have been used very freely and not, I think, with thorough understanding of what they mean. The original idea of Keith and his associates was that malignant hypertension is a condition associated with increased blood pressure, retinal changes and normal renal function, with death occurring from a cerebral accident. This condition, of course, is entirely different from malignant nephrosclerosis or necrotizing arteriolitis; the term "malignant hypertension" should be applied only to this devastating change in the arteries, and not to every case of markedly high blood pressure, as is the habit of many physicians. A much greater insight into the diagnosis of the retinal lesions can be obtained if one can accurately correlate the ophthalmoscopic picture with the changes that occur in the blood vessels rather than with a clinical syndrome, such as diabetes, hypertension or glomerular nephritis.

DR. W. W. HERRICK: If I have any suggestion to make it is that a study of this sort be extended to include the earlier stages of this condition. Those present have in this study heard of the fundus changes characteristic of the late or terminal stages of arterial disease, of diabetes and of nephritis but nothing about the changes which mark the earlier stages of such diseases. I believe this is a great field for cooperative work among the internists and ophthalmologists.

Roughly, for clinical purposes, one can classify the diseases of the kidney and the arteries under discussion as the nephroses, the nephritides and the primary arterial diseases. Of the nephroses the most common is that of pregnancy. In that condition one sees spasticity of the arteries, a narrowing in their caliber, which often varies from day to day. Soon thereafter there is edema of the general retinal field, perhaps including the optic nerve head. Later one sometimes sees hemorrhage, very rarely patches of exudate and occasionally retinal separation, which, in our experience at the Sloane Hospital for Women, has a fairly good prognosis. Conditions of that sort, usually accompanied by hypertension, clear up with delivery, but in the subsequent years at least 40 per cent of the patients will come to have essential hypertension and the ophthalmoscopic changes associated with that disease. In essential hypertension, which internists consider a primary functional disorder characterized by a rise in blood pressure with secondary changes in the small vessels, with a high systolic and a correspondingly high diastolic pressure, with death resulting most often from cerebral or cardiac changes and rarely from renal insufficiency secondary to renal sclerosis—in this most common of arterial diseases, there is first of all a spastic condition of the retinal vessels: an arteriovenous constriction and distention of the veins. Only when the condition is more advanced does one see an exudate. This is most likely to be of the medium-sized grayish patch type and more rarely of the silver-white dot type. In the later stages there is hemorrhage. That is about as far as essential hypertension in the average patient goes in retinal changes unless there is reduction of the renal circulation to the point of renal failure. Then one sees papilledema, large amounts of yellowish exudate, most often in the macular region, with large hemorrhages and sometimes thrombosis. These are also the changes which one finds in patients with malignant

hypertension, which practically always ends in renal failure. It may be that this is a separate disease, since its pathology, as described by Dr. Mosenthal, is quite different and distinct from that of benign essential hypertension. In malignant hypertension the changes are those of the most advanced arteriosclerosis plus papilledema and large masses of exudate.

I believe one should confine the term "nephritis" to the inflammatory processes in the kidney; that one should not include in the term the nephropathies secondary to arterial changes. One should make an attempt to differentiate primary vascular renal disease from primary inflammatory renal disease. One cannot always do that in the late stages of either process, but in the earlier phases, and with the knowledge of the history of the disease, it is often possible. It is of great interest that the retinal changes in patients with nephritis do not run parallel with the clinical picture. One may see advanced retinal changes with moderate nephritis. One sometimes sees uremic death with very little change in the retina. I have lately observed a case in my own ward at the Presbyterian Hospital, an example of embolic nephritis with uremic death and practically no ophthalmoscopic lesions. When one does encounter retinal changes with nephritis, they are apt to be dramatic—marked papilledema, great patches of yellowish-white exudate and hemorrhage, with a secondary and relatively late development of arteriosclerosis.

DR. A. J. BEDELL: It seems to me that until one has the correlation of photographic reproductions of the fundus clinical findings and pathologic observations one will continue to indulge in speculation. Nothing but photographs of the fundus will serve the ophthalmologist's purpose.

There is a widespread misunderstanding regarding hypertension and arteriosclerosis. Dr. Cohen, fortunately, stresses one phase of the latter, and I agree with him that practically all the changes that one finds in the fundus of a patient with diabetes are arteriosclerotic in origin, with the possible exception of the hard whitish-yellow granular exudates which appear in and about the macular region. This is substantiated in bedside practice, in which, with the exception of lipemia, no changes are ever found in the fundi of children with diabetes.

Dr. Cohen makes the further statement that it is difficult or impossible for the ophthalmologist to diagnose renal or arterial disease without the cooperation of the internist. This, I am sure, is good teaching and often proved.

DR. DAVID WEXLER: I am inclined to agree with Dr. Mosenthal that the distinction between the benign and the malignant phase of essential hypertension is largely a matter of definition. The difference is an anatomic one and depends on the speed and degree to which the arterioles are narrowed. For instance, one is tempted to apply the term "malignant" to that form of essential hypertension in which a person in his thirties or early forties, usually with compensated renal function, succumbs to a cerebral or cardiac accident. Strictly speaking, malignant hypertension is synonymous with arteriolonecrosis or malignant sclerosis of the kidneys. On the other hand, the benign form exhibits varying degrees of slowly progressive arteriosclerosis of the kidneys. In this form, renal function is adequate, except perhaps in the terminal stages.

In the fundus, perhaps the safest single criterion of malignant hypertension is papilledema; furthermore, it is proper to consider the hypertension with papilledema malignant in the presence of adequate or compensated renal function, as pointed out by Keith and Wagner and by Fishberg and Oppenheimer several years ago. A clinicopathologic study of twenty-nine patients with essential hypertension observed at the Mount Sinai Hospital together with Dr. I. Goldstein confirms this opinion. This group includes only those whose eyes were studied histologically. Sixteen had benign and thirteen malignant hypertension. In the group with the benign form, death in six was due directly or indirectly to cardiac failure; three died of cerebral hemorrhage, two of arteriosclerosis of the kidneys and the remainder of intercurrent disease (cancer and various postoperative complications). In all the patients with the malignant form who were available for study, death was attributable primarily to renal insufficiency, and the kidneys showed malignant nephrosclerosis. In the group with the benign form the optic disk was invariably normal, although in nine there was retinitis of some degree. Papilledema was a constant feature in those with malignant nephrosclerosis. It is interesting to note that all of those who suffered cerebral accidents who were studied were in the group with benign hypertension. In these, the kidneys showed slight arteriosclerosis and the disks were normal in the presence of moderately severe retinitis. There was no instance of a fatal termination due to a cerebral hemorrhage in the group with malignant nephrosclerosis.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

Annual Congress, London, England, April 2, 3 and 4, 1936

R. Foster Moore, F.R.C.S., *President*

Ophthalmia Neonatorum. Mr. J. D. Magor Cardell.

The commonest source of infection is in the infected material implanted as the baby's head traverses the birth canal of the infected mother. The mother or the attendant may easily infect the baby's eyes with soiled hands. In making an examination of the infant, the non-infected eye should be examined first. The purulent discharge may contain micro-organisms, which should always be sought in smear and culture. When the discharge is confined behind swollen lids, there is an adverse effect on the cornea; if excessive force is used in opening the lids, the cornea may be damaged, and if the lids are carelessly opened, the pus may spurt into the eyes of the attendant.

With regard to complications, corneal ulceration is most to be feared, and it should be remembered that the cornea is susceptible to slight trauma. The lacrimal gland is rarely involved. The duration of the disease depends on the severity of the infection, the health and degree of resistance of the child and the efficiency of the treatment. The prognosis is more favorable when the infection is slight and when

institutional treatment can be secured. It is less favorable in the presence of marasmus, prematurity, gastro-enteritis and syphilis.

The treatment carried out for the condition should consist in hourly irrigation, day and night, accomplished by two nurses, one to apply the treatment and manipulate the upper lid, the other to steady the child's body and to retract the lower lid. Drops used are silver nitrate, strong protein silver, mild protein silver, mercurochrome, acriflavine, liquid petrolatum, hydrogen peroxide and lactic acid.

DISCUSSION

DR. H. P. NEWSHOLME: Most people think of gonorrhea when ophthalmia neonatorum is mentioned, but bacteriologic methods inculpate other organisms beside the gonococcus. The medical practitioner is required to notify the local authority of infants with purulent discharge beginning within twenty-one days after birth. Though the rates of incidence vary, owing to the varying degrees of energy shown by those engaged in notification, there has been in Birmingham in the last few years a marked diminution in the number of cases of severely impaired vision. The decrease of gonorrheal ophthalmia in Birmingham may be the result of three main factors: (1) a decrease in the prevalence of gonorrhea, (2) more efficient treatment and (3) an improved midwifery service, with better antepartum and better natal care.

With regard to prevention, the more freely and accurately pregnant women are examined the better. The Central Midwives Board has insisted on regular antepartum care and prompt reference of any abnormality to practitioners for advice. A 1 per cent solution of silver nitrate is instilled by the midwife into the conjunctival sac after birth, and if there is a conjunctival discharge the case must be reported to the local authority.

DR. S. H. BROWNING: The diagnosis and treatment in any given case must depend on the bacteriologic observations. Without the aid of bacteriology even the most skilful clinician must experience diagnostic difficulty. Treatment by vaccines and serums is not satisfactory. In patients with bilateral involvement both eyes tend to be cured at the same time, thus appearing to suggest that the level of resistance of the body is raised or that local immunity has occurred in both eyes at the same time. The prospect of cure depends largely on the constitution of the child. Syphilitic children with ophthalmia neonatorum do badly. Only 48 per cent of syphilitic children recovered without loss of sight, while 81 per cent of nonsyphilitic children recovered without such loss. Recently, Pneumococcus has frequently been found associated with ophthalmia neonatorum.

PROF. M. MÁRQUEZ: Means of fighting ophthalmia neonatorum are often used to excess. The use of silver nitrate is the standard medication, but the use of the silver nitrate stick is to be strongly deprecated. Potassium permanganate, 1 part in 4,000, used twice or thrice daily, is valuable. I am not convinced as to the value of strong protein silver (protargol and similar preparations).

SIR JOHN PARSONS: I consider that statistics based on notification returns are practically valueless, because the returns report many condi-

tions which are not simple and cannot be placed under one heading. It would be a different matter if separate tables could be drawn up of the cases of known gonorrheal ophthalmia neonatorum of the benign form.

DR. A. J. BALLANTYNE, Glasgow: There is no noticeable diminution in the number of cases of ophthalmia neonatorum in the Glasgow returns, but in the average case the severity of the condition is very much less than formerly.

COLONEL HENRY SMITH: The one method of dealing with the infected conjunctival sac is to divide the outer canthus freely and allow free drainage. There is no risk of injuring the cornea.

MR. A. F. MCCALLAN: In Egypt gonococcic ophthalmia is very prevalent, but ophthalmia neonatorum is very rare. The reason for this has never been given. It may be that trachoma and pannus with its vascularization protect the cornea.

DR. SPENCE MEIGHAN: I wish to emphasize the need for repeated bacteriologic examinations if no gonococci are found at the first examination.

MR. FRANK JULER: In a large maternity hospital, among 44,357 live babies delivered, 180 had ophthalmia neonatorum, an incidence of 0.47 per cent. A considerable proportion of the mothers were very poor, and some of them were unmarried.

MR. CHARLES GOULDEN: It is often taught that if an eye with this disease comes under treatment with the cornea intact and the eye is then lost it must be the fault of the person treating the eye. I have always held that streptococcic infection is an exception to this generality, because whatever one does in cases of streptococcic ophthalmia the eye frequently is lost. When using silver nitrate on the eyes of a baby, it is important to neutralize the silver nitrate with sodium chloride, because the baby has no tears and so there is no natural admixture with sodium chloride such as one would have in the eye of an adult.

A NEOPLASM OF THE TEMPORAL FOSSA ASSOCIATED WITH PROPTOSIS OF THE CORRESPONDING EYE. SIR ARNOLD LAWSON.

A woman aged 54 shows a growth occupying the right temporal fossa, with proptosis of the right globe. About 1924, she noticed a fulness over the right temple. Examination shows a large elastic uniformly smooth tender swelling occupying the whole of the temporal region, without glandular enlargement, discoloration of the skin, enlargement of superficial vessels or sign of inflammation. There is no limitation of movement or diplopia. The pupils are equal, small and sluggish to light stimulus, and the fundus and the circulation are normal. The thyroid gland is not enlarged. The late Sir Anthony Bowlby thought that it was probably an inoperable fibrosarcoma involving the right orbit and recommended treatment with radium, which was given in the Radium Institute. There is a diffuse thickening of the right side of the skull, the sclerosis involving the bony orbit. Forty milligrams of radium element screened with 2 mm. of lead was applied to the region of the tumor on a number of occasions, extending over two years. The growth became softer and less prominent, but in the last two years there has been little change. It seems that the exophthalmos and the temporal

growth occurred as a curious coincidence, that they were not related to each other. The thyroid now seems to be more conspicuous than formerly. The visual field is normal, and the central color vision is excellent. The growth is evidently of a low type of malignancy.

EXPERIMENTAL HETEROGENEOUS CORNEAL GRAFTS. MR. J. W. TUDOR THOMAS.

This is an account of six experimental corneal grafts on rabbits, the transplanted tissue being obtained from the corneas of animals of another species. One of the grafts was taken from a human cornea and the other five from corneas of cats. In each case use was made of a graft having a shelving margin. Unlike successful homogeneous grafts, which remain transparent, heterogeneous grafts become vascularized. The blood vessels come from the iris or, by growth through the recipient cornea, from the limbus. The primary union of the graft tends to be incomplete, and some loss of tissues from the graft readily occurs. The epithelium is lost and is replaced by a growth of epithelium from the recipient cornea, commencing in much less than two months. The epithelium is supported by a subepithelial layer of new vascularized tissue, derived largely from the stroma of the recipient cornea. Behind the graft a layer of fibers is formed from the endothelium and deep corneal fibers of the recipient eye, and behind this there develops a new posterior elastic membrane which is less than one third of the thickness of the Descemet membrane of the recipient cornea. While the original epithelium of the graft is lost, Descemet's membrane remains. The original stroma of the graft can still be distinguished as isolated fibers in the subepithelial level, but in the posterior level it remains segregated, by the growth of new tissue around it, as a mass of poorly staining fibers, fairly homogeneous in appearance, with few nuclei and practically no blood vessels. These observations seem to indicate that the heterogeneous graft is tolerated but largely ostracized by the recipient eye and that therefore heterogeneous corneal grafts should not be used for corneal transplantation in man.

RADIUM NECROSIS OF THE CORNEA. MRS. PHILIPPA MARTIN.

The time of onset of corneal necrosis depends on the intensity of the irradiation. The earliest sign is a diminution of corneal sensation, the response to touch with cotton-wool being slower than in the normal eye. After a few weeks or months the cornea may lose its polish, and later small superficial ulcers may appear and coalesce to form a large superficial ulcer. Pain becomes severe when there is a punctate infiltration, and with true irradiation iritis the pain is severe. In all cases in which massive irradiation is proposed, the pupil should be dilated with a mydriatic before the treatment is commenced and its exact size noted. After irradiation the degree to which the pupil dilates under the same mydriatic is then an indication of the presence or absence of iritis. Protection of the eye by a metallic screen is dangerous, since the screen becomes a source of secondary irradiation.

Book Reviews

Detachment of the Retina; Operative Technique and Treatment. By J. Cole Marshall. Price, $2.75. Pp. 88. London: Oxford University Press, 1936.

This small book does not pretend to discuss the subject of retinal detachment completely. There is no consideration of the pathogenesis or even a description of the various clinical types of detachment. Marshall is wholly concerned with the treatment of detachment by modern operative methods. Four types of procedure are described—the cautery puncture of Gonin, the chemical method of Guist and of Lindner, the method of electrocoagulation of Weve and of Šafář and the use of electrolysis by Vogt and by von Szily. The descriptions include practical details observed by the author in most of the clinics where the procedures were first employed and details noted by him in his own operations.

Preparation for the operation is properly emphasized, the most important part of which is localization of the retinal tear that is usually present. This is mapped out in relation to various landmarks, and its position is recorded on a chart for use in the operating room. The charts published by Hamblin for this purpose, which seem convenient, are shown in illustrations.

Marshall feels that the procedure of Gonin, which most ophthalmologists have abandoned in favor of methods of electrocoagulation, possesses merits which will cause it to be used more frequently in certain cases, especially those in which there is a single small hole. He describes the combination of this method with scleral coagulation, which was employed by Gonin in his later operations. He seems to have had more experience with the method of Weve and Šafář, in which are employed both surface coagulation to the sclera and pins 1.5 mm. long which are inserted in the area already coagulated and with which the choroid in the area of the tear is coagulated. The importance of repeated observation of the fundus during the operation, to enable one to be sure of the relation of the coagulated areas to the tear, is emphasized. Weve's longer needles are employed for this purpose, when necessary. The illustrations showing the results of operation in illustrative cases are excellent.

In discussing electrolysis Marshall describes both the method employed by Vogt and that employed by von Szily, but has apparently failed to grasp von Szily's careful explanation of the rationale of his procedure. The method employed by von Szily depends on the application of both poles of the circuit to the eye at a short distance apart, so that the whole effect is confined to this small area. The effect is not that of heat, as with the methods of diathermy, but is a chemical effect due to the dissociation of hydrogen and hydroxyl ions in the tissues. Either both the poles are applied to the sclera or one is placed on the sclera while the other penetrates to the choroid. From the short

bibliography one misses some of the more important articles by the authors whose works were consulted. One misses also a studied comparison between the methods, especially as to the percentage of the successful results obtained with each, and conclusions as to the indications for the various methods in certain types of detachment. No summary of the author's own results is given.

SANFORD R. GIFFORD.

La radiographie en ophthalmologie. By E. Hartmann. Price, 120 francs. Pp. 280, with 391 illustrations. Paris: Masson & Cie, 1936.

This is the eighth of a series of works published under the auspices of the French Ophthalmologic Society. In format, quality of paper, reproduction of the numerous plates and in contents it comes up to the standards set by this series, which has provided a notable addition to the ophthalmologic literature of the past ten years. The illustrative roentgenograms were chosen from Hartmann's collection and from a large number sent by colleagues of various countries in response to his request.

The various methods of exposure are considered with regard to their advantages in demonstrating the parts of the orbit, with illustrative roentgenograms. By the method of Brunetti, for instance, which seems to be little known in America, perfectly clear outlines of both sphenoidal fissures are shown on one plate. This is achieved by placing the patient's brow on the plate, while the tube is employed at an angle of 15 degrees to the right and left sides, the opposite half of the plate being protected by lead during the exposures.

The visibility of calcification in the lens, vitreous and choroid is described and illustrated. Calcification of the cerebral vessels in the region of angiomas associated with von Hippel's disease is illustrated, and numerous illustrations of calcification of the internal carotid and ophthalmic arteries with evidence of pressure on the optic nerve are shown.

The detection and localization of intra-ocular foreign bodies are fully discussed. It is worth remembering that after removal of a metallic foreign body a sufficient deposit of metallic salts may be left to cast a definite shadow suggesting that a foreign body is still present. Fragments of wood and glass are illustrated by views taken under favorable conditions. The procedure of Wilder, who placed a piece of the same glass, in cases in which it was available, on the cheek to determine whether it was radiopaque, might well have been mentioned. Six groups of methods employed for localization are discussed, including the "bone-free" method of Vogt, the geometric methods, among which that of Sweet is best known, and the methods depending on making the globe visible by the injection of a radiopaque substance into Tenon's capsule. Of the "simple methods," the use of a contact glass with a radiopaque mark corresponding to the limbus, according to the method of Wessely, and the method of Velter, in which beads of lead are fastened to the limbus above and below, are among the most practicable. The latter method is almost the same as that of H. Gifford, who employed pieces of silver wire in the same way.

A great variety of tumors of the orbit are illustrated, as are a variety of fractures involving the orbit and the optic foramina. Thirty different methods of exhibiting the optic foramen have been proposed, and these, including the author's own method, are discussed.

Roentgenography of the lacrimal passages with the aid of opaque fluids is discussed and illustrated, as are the bony changes in cases of sinus disease, cerebral tumors, tumors of the hypophysis and intracranial hypertension.

The book should greatly aid both the ophthalmologist and the roentgenologist in obtaining the most useful views and in properly interpreting them.

SANFORD R. GIFFORD.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.
Place: Cairo. Time: December 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.
Place: Oxford. Time: July 22-24, 1936.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. R. Foster Moore, 53, Harley St., London, 1.
Secretary: Miss Ida Mann, 7, Wimpole St., London, 1.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: July 6-8, 1936.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. John Green, 3720 Washington Blvd., St. Louis.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Kansas City, Mo.: Time: May 11-15, 1936.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: New York. Time: Sept. 26 to Oct. 3, 1936.

American Ophthalmological Society

President: Dr. L. S. Greene, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 1-3, 1936.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City, Utah. Time: Spring, 1937.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.
Place: Seattle. Time: January 1937.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. W. R. Fringer, 228 S. Main St., Rockford, Ill.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.
Place: Savannah. Time: May 12-15, 1936.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Edwin Cob, 307 Masonic Temple, Marshalltown.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Marshalltown. Time: September 1936.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. E. Binger, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Place: St. Paul. Time: May 1936.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hosp., Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Secretary: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Burton W. Fassett, Geer Bldg., Durham.
Secretary-Treasurer: Dr. Casper W. Jennings, 332 N. Elm St., Greensboro.
Place: Durham. Time: October 1936.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.
Place: Jamestown. Time: May 1936.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Emory Hill, 501 E. Franklin St., Richmond.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.
Place: Richmond. Time: May 1936.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William O. Martin Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. A. V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Robert B. Blue, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. F. W. Thomas, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Newton, Mercantile Bldg., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. T. L. Goodman, 602 W. 10th St., Forth Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. John H. McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. R. M. Hargrove, 1202 Esperson Bldg., Houston, Texas.
Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. William A. Boyce, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Hilmar G. Martin, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer. Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member in alphabetical order.
Secretary: Dr. C. W. Rucker, 527 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. W. J. Blackburn, 663 Reibold Bldg., Dayton, O.
Secretary-Treasurer: Dr. F. J. Driscoll, 986 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Kate S. Zerfoss, 119, 7th Ave., N., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LaGrand H. Hardy, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. E. C. Reynolds, 657 Main Ave., Passaic, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Robert H. Courtney, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Carl T. Eber, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Greene, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.